SPECIAL COLLECTIONS

UNIVERSITY OF ALBERTA LIBRARY

REQUEST FOR DUPLICATION

I wish a photocopy of the thesis by

Borysyk, Lawrence M. (author)

entitled The Effect of Active & Passive Warm-up.

The copy is for the sole purpose of private scholarly or scientific study and research. I will not reproduce, sell or distribute the copy I request, and I will not copy any substantial part of it in my own work without permission of the copyright owner. I understand that the Library performs the service of copying at my request, and I assume all copyright responsibility for the item requested.

Date	Name and address	Pages copied	Signature
Feb 3/83	LORI BRAGG 11715 28 AVE Edm.	1,2,3,4,44	Lori Bragg

F. 236

September 1975

THE UNIVERSITY OF ALBERTA

THE EFFECT OF ACTIVE AND PASSIVE WARM UP ON PLASMA FREE FATTY ACID CONCENTRATION

by

LAWRENCE M. BORYSYK

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF SCIENCE

DEPARTMENT OF PHYSICAL EDUCATION

EDMONTON, ALBERTA

SPRING, 1977

DEDICATION

To Father Time, my many advisors over the years, and Sue.

ABSTRACT

The effect of warm-up [elevation of deep body temperature (B_t) 0.5°C] on plasma FFA and lactate concentration during the warm-up and in a subsequent 30 min exercise (CT) at 60% $\dot{V}O_2$ max was investigated on 11 male subjects. Prior to the CT subjects underwent either active (40% $\dot{V}O_2$ max) ergometer exercise (T_e); passive heating in a sauna (T_s); or received no warm-up (T_c). Plasma FFA and lactate concentration did not change during either warm-up (T_s or T_e) condition. T_s, T_e or T_c had no effect on plasma FFA during the CT, however, in recovery, significant increases were observed after T_e. Immediate significant increases in lactate during the CT occurred when preceeded by T_c and T_s but not with T_e. It was concluded that the warm-up alone had no effect on plasma FFA during the warm-up or during the subsequent CT. The T_e condition resulted in significantly lower lactates than T_s or T_c throughout the CT as well as significantly higher plasma FFA in recovery.

ACKNOWLEDGEMENTS

The author wishes to thank Dr. H. A. Wenger, Chairman of the thesis committee, and Drs. A. Quinney and L. Wang, committee members, for their contribution to this study.

In addition I wish to express special gratitude to Dr. Dave Secord for his insight and wisdom, and to Dr. Phil Gardiner for his energy and help throughout the experiment. And, special thanks to Susan Cary for her patience.

TABLE OF CONTENTS

Page

INTRODUCTION 1

Definition of Terms 5

METHODOLOGY 6

Subject Selection and Orientation 6
Maximal VO_2 Test 7
Respiratory Gas Analysis 7
Experimental Conditions 8
Heart Rate and Deep Body Temperature 12
Blood Sampling Procedure 12
Statistical Procedure and Experimental Design 13

RESULTS 15

DISCUSSION 33

CONCLUSIONS 44

REFERENCES 45

APPENDICES

A. Review of Literature 53

B. Tables of Analysis of Variance and Newman-Keuls Tests. 70

LIST OF TABLES

Table Page

1 Characteristics of Subjects 16

2 General Test Data 16

LIST OF FIGURES

Figure Page

1 Time Plot Marked in 5 Minute Intervals for Control (Figure 1a), Exercise and Sauna (Figure 1b) Treatment Conditions 10

2 Comparison of Plasma Free Fatty Acid (Means ($\pm S_{\bar{e}}$)) for Control (o), Exercise (Δ), and Sauna (□) Treatment Conditions During Rest, Exercise, and Recovery 17

3 Comparison of Changes in Plasma Free Fatty Acid from Rest Through Recovery for Control (o), Exercise (Δ) and Sauna (□) Treatment Conditions 19

4 Comparison of Lactate Means ($\pm S_{\bar{e}}$) for Control (o), Exercise (Δ) and Sauna (□) Treatment Conditions During Rest, Exercise and Recovery 22

5. Comparison of Respiratory Quotient Means ($\pm S_{\bar{e}}$) for Control (o), Exercise (Δ) and Sauna (□) Treatment Conditions During the Criterion Exercise 24

6 Comparison of Deep Body Temperature Means ($\pm S_{\bar{e}}$) for Control (o), Exercise (Δ) and Sauna (□) Treatment Conditions During Rest, Exercise and Recovery 27

7 Comparison of Oxygen Consumption Means ($\pm S_{\bar{e}}$) for Control (o), Exercise (Δ) and Sauna (□) Treatment Conditions During Rest, Exercise and Recovery. 31

INTRODUCTION

Carbohydrates and fats have been established as the main energy sources for muscular activity (Newshome and Start, 1973). The extent of metabolic mixing of these two fuels depends not only on the intensity and duration of the exercise, but also on the subject's state of fitness (Pruett, 1970; Saltin, 1973). Although it was long believed that carbohydrate was the only fuel for energy generation (Astrand and Rodahl, 1970), the importance of lipids during certain intensities and durations of muscular exercise has been substantiated within recent years (Carlson, 1967; Gollnick, 1969; Havel, 1971; Paul and Holmes, 1975; Saltin, 1973). Pruett (1970) found that even at extreme exercise intensities where the primary energy source is carbohydrate, probably ten percent of the energy is delivered from lipid stores. During moderate exercise in which there is little or no increase in blood lactate level, the utilization of fat as a fuel may provide 25-90% of the energy for the aerobically working muscle (Paul and Holmes, 1975; Havel, 1971). In moderate exercise, fuel substrate appears to be in a transitory state (Astrand and Rodahl, 1970; Pruett, 1970), utilizing glycolytic resources at the beginning, while transferring to lipid metabolism as steady state is achieved.

Free Fatty Acids (FFA), once delivered to the cell membranes, are readily taken up and oxidized during exercise (Carlson and Pernow, 1959; Friedberg et al., 1963). Due to its rapid turnover rate (Gordon and Cherkis, 1956), the levels of plasma FFA appear to be controlled by the rate at which FFA is mobilized from adipose stores (Paul, 1975). The rate of FFA removal was found to be the result of a mass action effect of FFA concentration in the plasma (Eaton and Steinberg, 1961;

Fritz, 1957; Friedberg et al., 1960; Briefberg et al., 1963). Armstrong (1961, p. 9) states:

> Under a variety of conditions, changes in FFA concentration are brought about by changes in FFA production rate and that changes in FFA uptake are simple mass action effects of changes in FFA concentration.

He (Armstrong, 1961) noted that the concentration of FFA in the plasma may only be indicative of the rate at which FFA is being taken up by the active muscle. FFA uptake may be facilitated, thus lowering FFA concentration in the plasma, even though the rate of FFA production were to remain unchanged or even increased. Subsequent experiments have shown that, during exercise, concentrations of FFA decrease due to an increased fractional turnover rate (Friedberg et al., 1963; Carlson and Pernow, 1961; Bruce et al., 1961; Cobb and Johnson, 1963). After a duration of about 15 minutes, plasma FFA start to increase due to increased mobilization (Basu et al., 1960; Carlson and Pernow, 1961; Friedberg et al., 1963; Havel et al., 1963), with the highest levels reached after exercise (Cobb and Johnson, 1963; Taylor et al., 1971). Rates of FFA mobilization and subsequent uptake by the muscle are higher during exercise that at rest at any equivalent FFA level (Issekutz et al. 1964; Paul, 1970).

A reciprocal relationship appears to exist between FFA and blood glucose levels so that, when blood glucose is high, FFA levels are low; and when FFA levels are high, glucose is normal or may be slightly depressed (Gollnick, 1969). Elevation of blood lactate exerts an inhibitory effect on the release of FFA from adipose tissue (Cobb and Johnson, 1963; Issekutz et al., 1965; Miller et al., 1963; Boyd et al., 1974).

Studies on the effect of external heating on normal subjects are not uncommon (Sancetta et al., 1964; Bell et al., 1965; Carlesten et al., 1961; Goldman et al., 1965; Eisalo, 1956; DeVries et al., 1960); however, only a few to date have examined FFA levels under thermal stress (Taggart et al., 1972; Britton et al., 1974; Eddy et al., 1976). Eddy et al. (1976) have reported significant (p .05) increases in FFA levels in subjects who dehydrated with heat exposure as opposed to subjects who replenished fluids while exposed to heat. Their (Eddy et al., 1976) procedure using a climatic chamber and clothed subjects differed radically from the way in which Britton et al. (1974) and Taggart et al. (1972) exposed their subjects. These latter two studies reported small, statistically insignificant increases in plasma FFA levels in nude subjects exposed briefly to actual sauna conditions.

Investigations as to whether a passive warm-up in a sauna affects FFA mobilization on subsequent exercise do not exist. It is known that increased heat production associated with muscular exercise leads to a rapid temperature rise in working muscles and a more gradual increase in body core temperature (Robinson et al., 1965). Barcroft and King (1910-11) noted that the dissociation of oxygen from hemoglobin is more complete at higher muscle temperature, thus enhancing oxygen supply during work. This would enhance the capability for beta-oxidation of fatty acids during the high temperature conditions. Astrand and Rodahl (1970, p. 524) sum up the benefits of increasing body temperature through warm-up stating:

> ...metabolic processes in the cell can proceed
> at a higher rate, since these processes are
> temperature dependent.

Through such arguments, warm-up has been deemed essential for optimal

performance in sport. Martin (1975, p. 146), however, states that "actual physiological data on the question has been indeterminate." In assessing the effects of warm-up on both aerobic and anaerobic energy transformations in treadmill running of 1.5 and 5 minute durations, he (Martin, 1975) showed an 11% increase in heart rate and an 8% increase overall in oxygen consumption when warm-up was used. Running following warm-up resulted in 25% lower lactate production, as well as 3 to 4°C higher gastrocnemius muscle temperatures.

It was, therefore, of great interest and practical value to study whether warm-up tends to promote plasma FFA concentration and thus enhance the availability of substrates early in the exercise, perhaps improving performance in moderate exercise of long duration.

Specifically, this study tried to answer the following questions:

1. Does a passive increase in deep body temperature, using a sauna, enhance plasma FFA concentration?
2. Does a passive increase in deep body temperature in a sauna differ from an active increase in deep body temperature through exercise, with respect to plasma FFA concentration?
3. If each warm-up, active and passive, and a control condition are immediately followed by exercise of moderate intensity, will there be a differential change in plasma FFA concentration during the exercise and a change in metabolic emphasis as indicated by shifts in the respiratory quotient (RQ)?
4. Does the FFA level bear any relationship to the measured oxygen consumption taken at similar intervals during the moderate intensity exercise condition?

Definition of Terms

Deep body temperature: refers to the internal temperature (°C) of the body as measured by a rectal thermistor to a depth of 7-10 centimeters.

Passive warm-up: refers to the procedure used to elevate the subject's deep body temperature 0.5 ± 0.1°C. via external means of a sauna.

Active warm-up: refers to the procedure used to elevate the subject's deep body temperature 0.5 ± 0.1°C. via exercise at a workload equivalent to 40% of the subject's maximal oxygen consumption.

Maximal oxygen consumption ($\dot{V}O_2$ max): The point at which oxygen consumption per minute has attained its maximum in an exercise to exhaustion. (Volume expressed in liters of oxygen per minute or as a function of body weight as in ml/kg/min.)

FFA turnover: refers to the differential rate of release into and the rate of uptake from the plasma of free fatty acids.

FFA mobilization: refers to the process by which adipose cells are stimulated to release FFA into the circulation.

METHODOLOGY

The purpose of this study was to investigate the effects of an active (exercise) and a passive (sauna) warm-up on plasma FFA concentrations during an aerobic work bout. All subjects experienced an active, passive, and no warm-up condition prior to a criterion exercise test at 55-65% of their maximal oxygen consumption ($\dot{V}O_2$ max). Oxygen consumption, heart rate, and blood samples for FFA and blood lactate analysis were taken at selected intervals during rest, warm-up, and exercise.

Subject Selection and Orientation

Eleven male volunteers, ranging in age from 19 to 30 years, were chosen for this study. Those selected were either current students at the University of Alberta or players in training with a City of Edmonton Rugby team. Table 1 lists the personal data of the subjects. Subject selection was based on an initial $\dot{V}O_2$ max test on a bicycle ergometer. A $\dot{V}O_2$ max of 45 ml/kg was chosen as a baseline, and subjects achieving less than this value were eliminated from the study. Subjects were asked to maintain their current activity pattern and diet throughout the experimental period. In addition to 2 maximal tests, one at the beginning and the other at the end of the study, all subjects were asked to report for three additional 2-hour sessions. With the exception of the maximal tests, subjects reported to the laboratory between 7 am and 11 am on test days in a post-absorptive state. The testing sessions for each subject were scheduled approximately 1 week apart and all testing was completed within 12 weeks.

Maximal $\dot{V}O_2$ Test

$\dot{V}O_2$ max for each subject was determined on the bicycle ergometer following the procedure described by Cumming (1972). Prior to each test, the subjects were weighed on a medical scale and then connected to an electrocardiogram. Lead placement was standardized for heart rate monitoring only. Initial resistances of 2 or 2.5 kp were chosen depending on the size and the activity level of each subject. Resistance for the second work load was increased by 1 kp. At the completion of the two 6-minute submaximal loads, progression toward $\dot{V}O_2$ max began with increases of 0.5 kp per minute until the subject was exhausted. Pedal frequency was maintained at 50 revolutions per minute with the aid of a metronome, and verbal encouragement was given to maintain the frequency at the high work loads. The number of pedal revolutions was monitored by connecting an electric counter in series with the microswitch on the ergometer and a Gralab timer. Each subject was instructed to indicate by a hand signal when approximately one more minute of exercise could be maintained; however, completion of the minute was not necessary as oxygen consumption was being monitored continuously. Heart rate was recorded every two minutes during the submaximal levels, and every minute thereafter to fatigue. A Sanborn 500 Viso-cardiette portable electrocardiograph was used for monitoring heart rate. The laboratory where the exercise tests were conducted was maintained at a temperature of 20-21 C; however, humidity was not controlled. Barometric pressure and temperature were recorded at each exercise session.

Respiratory Gas Analysis

An on-line continuous sampling procedure was employed for respiratory gas analysis. A Beckman physiograph with AC-DC couplers

amplified and recorded the following parameters: volume of inspired air, percent expired oxygen, and percent expired carbon dioxide.

Recording of inspired air volume ($\dot{V}_I$) - The subject inspired room air through a Collins triple-J valve in series with a Parkensen-Cowan volumeter. A potentiometer within the volumeter converted the mechanical recording of each inspiratory volume to an electrical signal recorded on the Beckman recorder.

Analysis of expired air - The subjects' expired air was passed through a 6 liter baffled mixing box. A sample of the expired air could be drawn off from the mixing box to be analyzed for oxygen (O_2) and carbon dioxide (CO_2) content. The length of the flexicoil plastic hose connection the volumeter to the Collins triple-J valve and then to the mixing box was kept to a minimum to avoid excessive dead space. A Godart capnograph and a Beckman oxygen analyzer, Model F3, were connected in series with short plastic tubing. The continuous sample flow first passed through the CO_2 analyzer, then the O_2 analyzer. A time delay of approximately 5 seconds existed from volume recording to the CO_2 analysis display on the Beckman physiograph, and a further 10 seconds from CO_2 analysis to O_2 recording. This delay was considered in all calculations.

Calibration of O_2 and CO_2 analyzers - The two analyzers were calibrated prior to each testing session. Both analyses were done using room air and a known calibration gas. The calibration gas itself was checked periodically at the University Hospital Blood Gas Laboratory.

Experimental Conditions

Treatment conditions - Subjects performed each of the three treatment

conditions listed below in a random order.

Passive sauna warm-up (T_s): Each subject entered the sauna and remained inside until a deep body temperature increase of 0.5 ± 0.1 C was achieved.

Active warm-up using muscular exercise (T_e): Each subject pedalled at 40-50% of his $\dot{V}O_2$ max until a deep body temperature increase of 0.5 ± 0.1 C was achieved. Heart rates and deep body temperatures were recorded at 1.5 - 2 minute intervals during the exercise. As each subject approached the required 0.5 C increase, the submaximal oxygen cost was measured.

No warm-up (T_c): Each subject continued to sit passively for 15 minutes after an initial rest phase.

Figures 1a and 1b outline the time course for treatments T_s, T_e, and T_c. Duration of treatments T_s and T_e varied depending upon the 0.5 ± 0.1 C increase in deep body temperature. Treatment T_c was standardized at 15 minutes. Blood sample 3 was drawn approximately 5 minutes after the cessation of each treatment condition (Figure 1).

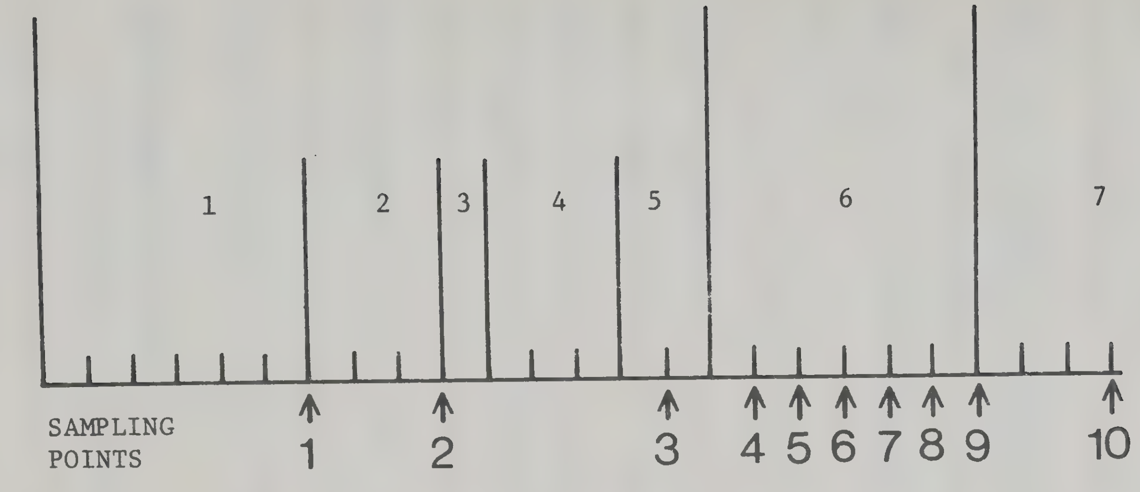
Criterion exercise test (CT) - The criterion exercise test constituted cycling at 55-65% of each subject's $\dot{V}O_2$ max for 30 minutes. Workloads corresponding to 55-65% of the subject's $\dot{V}O_2$ max were obtained using the Astrand and Rhyming nomogram (Astrand, 1960). Resistance settings of 3 and 3.5 kp were used with a pedal frequency of 50 rpm. The criterion exercise test followed each treatment condition by approximately 10 minutes (see Figure 1) with the same resistance setting over the three tests. A continuous recording of volume of inspired air ($\dot{V}_I$), as well

FIGURE 1

Time Plot Marked in 5 Minute Intervals for Control (Figure 1a), and Exercise and Sauna (Figure 1b) Treatment Conditions.

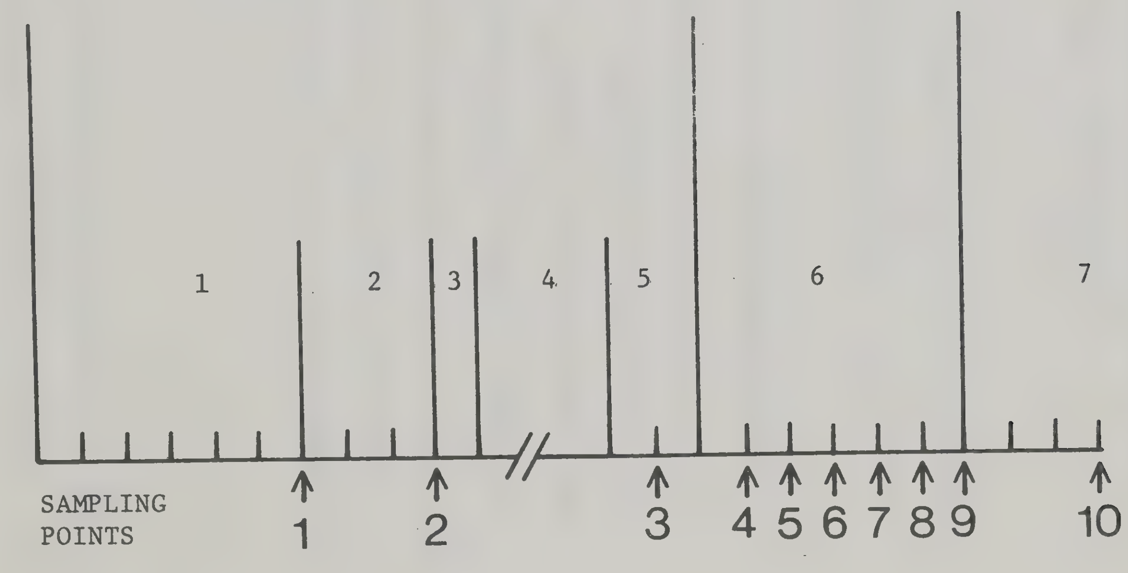
1a.

1. Preparation
2. Rest
3. Transfer
4. Rest (CONTROL)
5. Exercise Preparation
6. Criterion Exercise
7. Recovery



1b.

1. Preparation
2. Rest
3. Transfer
4. Warm-up (SAUNA or EXERCISE)
5. Exercise Preparation
6. Criterion Exercise
7. Recovery



as analysis of expired air permitted calculation of the subject's $\dot{V}O_2$ at any point during the 30 minutes of cycling. Six points corresponding to each of the blood withdrawal times were chosen for gas analysis (Figure 1). At the completion of the 30 minutes of exercise, gas analysis was terminated and the subject remained on the bicycle for a 15 minute recovery period followed by a final blood sample.

Heart Rate and Deep Body Temperature

The electrocardiogram was used to record heart rate at rest and at every 1.5 to 2 minutes throughout the 30-minute criterion test (CT; see page 10).

A battery-powered portable recorder for deep body temperature (YSI Tele-Thermometer, model 44TF) was calibrated prior to each test with a standard mercury thermometer. A semi-rigid thermister was inserted by each subject to a depth of 7 to 10 cm. into the rectum. Recordings were taken visually from the meter dial at similar sampling intervals to heart rate throughout the 30-minute criterion exercise test. For treatments T_c and T_e, both heart rate and deep body temperature were recorded at 1.5 and 2 minute intervals, whereas, for treatment T_s, only core temperature was recorded.

Blood Sampling Procedure

Ten blood samples were drawn at designated times (Figure 1). The blood samples were withdrawn by a registered nurse using a 21 gauge catheter (intermittent vein infusion set, TRQVENOL-CODE 2C0078) inserted into the brachial or anticubital vein of either arm. Coagulation of blood in the tubing (8.9 cm in length) and needle (1.9 cm) was prevented by using a solution of heparin and normal saline [0.1 ml heparin

(1:1000) in 100 ml normal saline]. After each blood sample was drawn, 1-2 ml of the anticoagulant solution was injected into the catheter and then withdrawn just prior to the next blood sampling. Approximately 5 ml of blood was drawn at each sample interval, 1 ml of which was pipetted immediately into 2 ml of an 8% perchloric acid solution in preparation for subsequent lactate analysis. The remainder of the blood sample, as wellas the deproteinized solution, was then refrigerated until test completion at which time all blood samples and lactate preparations were centrifuged for 10 minutes at 3000 rpm. The Sigma Kit method (Sigma Chemical Company, 1968) was used to determine lactate concentration and a colorimetric method to assay the serum for FFA (Pinelli, 1973).

Statistical Procedure and Experimental Design

Design - The experimental design utilized was a 10 x 3 factorial design with repeated measures on factor B (Winer, 1971). The 10 levels of factor A were:

1 - Rest 1 (R_1)
2 - Rest 2 (R_2)
3 - Post Treatment (PT)
4 - Exercise, 5 minutes (Ex5)
5 - Exercise, 10 minutes (Ex10)
6 - Exercise, 15 minutes (Ex15)
7 - Exercise, 20 minutes (Ex20)
8 - Exercise, 25 minutes (Ex25)
9 - Exercise, 30 minutes (Ex30)
10 - Recovery, 15 minutes (REC)

The 3 levels of factor B were:

1 - Control
2 - Exercise
3 - Sauna

The order of completion of factor B was randomized for each subject.

Statistical Procedure - A two-way analysis of variance with repeated measures was utilized (ANOV 23). If significant F ratios were obtained, the data was plotted and a modified one-way analysis of variance was performed to test simple main effects. The modified one-way analysis of variance made provision for a pooled error term to be used in computing F ratios. When F ratios for simple main effects were significant, a Newman-Keuls test was used as a comparison between means.

A 't'-test for correlated observations (ANOV 12) was used to test for significant differences between pre- and post-maximal $\dot{V}O_2$, maximal heart rate, maximal workload, and body weight.

RESULTS

Characteristics of subjects in this study are given in Table 1. Tables 2 and 3 give general data means and standard error (S_e). Paired 't' tests (ANOV 12) were conducted on maximal workload, maximal heart rate, maximal oxygen consumption, and weight obtained during the initial and final maximal exercise tests to discover whether changes occurred over the 12-week period. No significant changes ($p > .05$) were observed.

The means and $S_{\bar{e}}$ for FFA at each sampling point for each treatment condition are present in Figure 2. No significant ($p > .05$) "A" main effects (sampling times) were seen (Table 4) indicating that for each treatment condition, FFA levels did not change significantly from rest through recovery. Significance ($p < .05$) was shown, however, for "B" main effect (treatment groups). With a subsequent one-way analysis of variance, significance was indicated for the post treatment (i.e. 5 minutes after either warm-up or control conditions) and recovery (i.e. 15 minutes post exercise) sampling intervals. To ascertain which of the means at each sampling time were significantly different from each other, a Newman-Keuls test for the comparison of means was performed. Although there was no significant difference ($p > .05$) between means of the post treatment sample, subjects tended to exhibit higher FFA levels after T_e and T_s as compared to T_c (Figure 3). In recovery, the exercise warm-up treatment condition showed significantly greater FFA levels ($p < .05$) than the control treatment condition (Table 5).

TABLE 1

CHARACTERISTICS OF SUBJECTS

subject	age (years)	weight (kg) initial : final	$\dot{V}O_2$ max (liters) initial : final
P.B.	20	88.6 : 84.8	4.07 : 4.87
D.A.	22	77.5 : 77.7	4.19 : 4.03
J.S.	26	89.5 : 89.5	4.57 : 4.70
G.W.	22	72.0 : 70.4	3.31 : 3.32
D.O.	19	69.8 : 70.7	4.57 : 4.59
R.M.	22	70.7 : 74.5	4.04 : 4.13
V.P.	20	77.3 : 77.3	4.00 : 3.22
S.S.	22	68.6 : 70.0	3.93 : 3.56
L.B.	30	66.8 : 66.4	4.07 : 4.15
B.C.	26	71.8 : 69.3	3.79 : 3.52
E.B.	28	70.0 : 70.5	3.76 : 3.82

TABLE 2

GENERAL TEST DATA

variable		mean	S_e
Workload (kpm/min)	Criterion test	1002.3	25.33
	Exercise warm-up	702.3	25.33
Sauna Time	(Min.)	15.6	1.12
Exercise Time	(Min.)	22.8	1.47
Sauna Temperature (°C)	Dry bulb	75.4	0.10
	Wet bulb	32.3	0.06
$\dot{V}O_2$ (L/min)	Criterion test	2.44	0.64
	Exercise warm-up	1.83	0.07
% $\dot{V}O_2$ (A or B/max)	A = Criterion test	60.82	1.83
	B = Exercise warm-up	45.74	1.94

FIGURE 2

Comparison of Plasma Free Fatty Acid Means ($\pm S_{\bar{x}}$) for Control (o), Exercise (△), and Sauna (□) Treatment Conditions During Rest, Exercise and Recovery

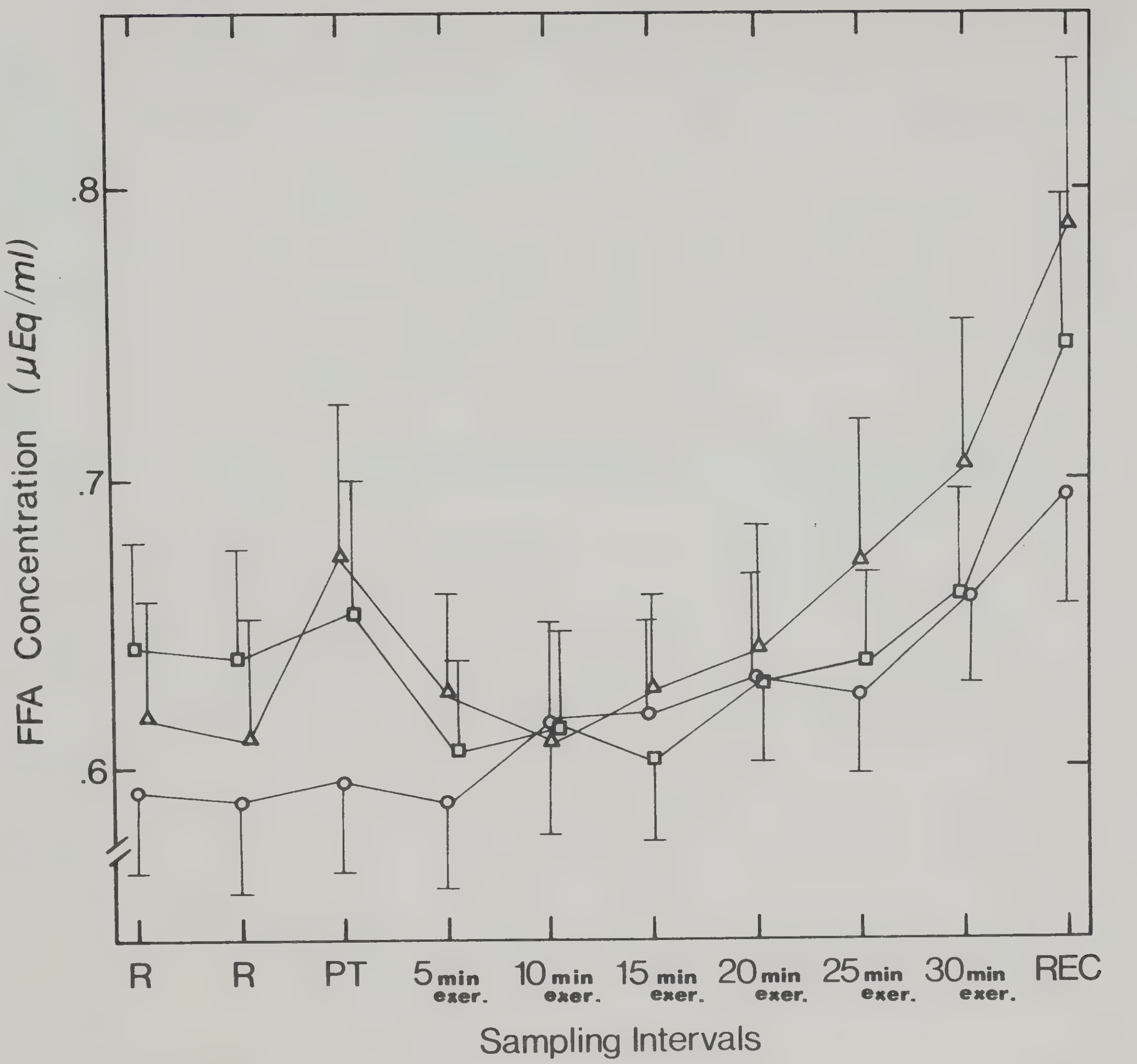
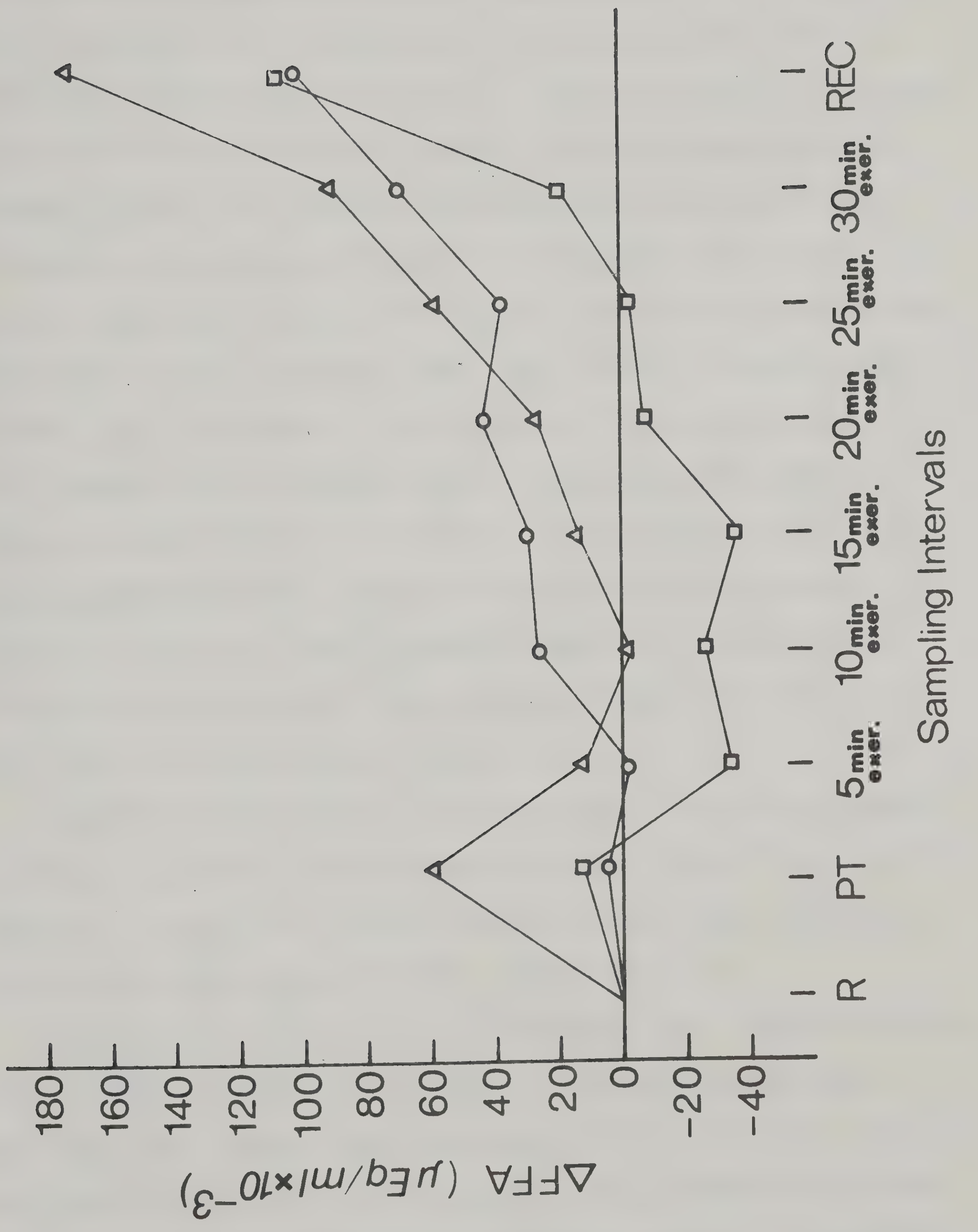

.8
.7
.6
FFA Concentration (μEq/ml)
R
R
PT
5 min exer.
10 min exer.
15 min exer.
20 min exer.
25 min exer.
30 min exer.
REC
Sampling Intervals

FIGURE 3

Comparison of Changes in Plasma Free Fatty Acid from Rest Through Recovery for Control (o), Exercise (▲) and Sauna (■) Treatment Conditions.


ΔFFA (μEq/ml×10^{-3})
180
160
140
120
100
80
60
40
20
0
−20
−40
R
PT
5 min exer.
10 min exer.
15 min exer.
20 min exer.
25 min exer.
30 min exer.
REC
Sampling Intervals

The means and $S_{\bar{e}}$ for lactates at each sampling period throughout each treatment condition are presented in Figure 4. Significance ($p < .05$) for both main effects (i.e. sampling times and treatment conditions) as well as the interaction of the two was shown (Table 6). Subsequent one-way analysis of variance indicated significant increases of lactate for each treatment condition from rest through recovery. Significant ($p < .05$) treatment effects were shown for the 10 minute through 30 minute exercise samples. In both the T_c and T_s groups, the lactate levels were significantly higher in all criterion exercise samples than in either of the two rest samples or post treatment sample. However, only the sample at the 30 minutes of exercise was higher than the resting or post treatment samples in the group which underwent an exercise warm-up (Tables 7, 8, and 9). Recovery samples for T_s and T_c were significantly lower than all the preceeding exercise samples. Except for the first 5 minute exercise samples for the group using exercise as warm-up, the 15 minute recovery sample was significantly lower than the last 5 exercise samples.

Analysis for any treatment effects showed significant differences across T_c, T_e, and T_s at the 10 minute through to the 30 minute exercise samples. At each of the points, lactate during T_e were significantly lower from lactates during T_c and T_s (Tables 10 to 14).

The means and $S_{\bar{e}}$ for R.Q. at each sampling point within each treatment condition are presented in Figure 5. No significance (Table 15) was shown for 'A' main effects (sampling times). Significance ($p < .05$) was shown for 'B' main effects treatment groups, however, a subsequent one-way analysis of variance across treatments indicated no significant ($p > .05$) differences. Each treatment condition showed a statistically

FIGURE 4

Comparison of Lactate Means ($\pm S_{\bar{e}}$) for Control (o), Exercise (Δ) and Sauna (□) Treatment Conditions During Rest, Exercise, and Recovery

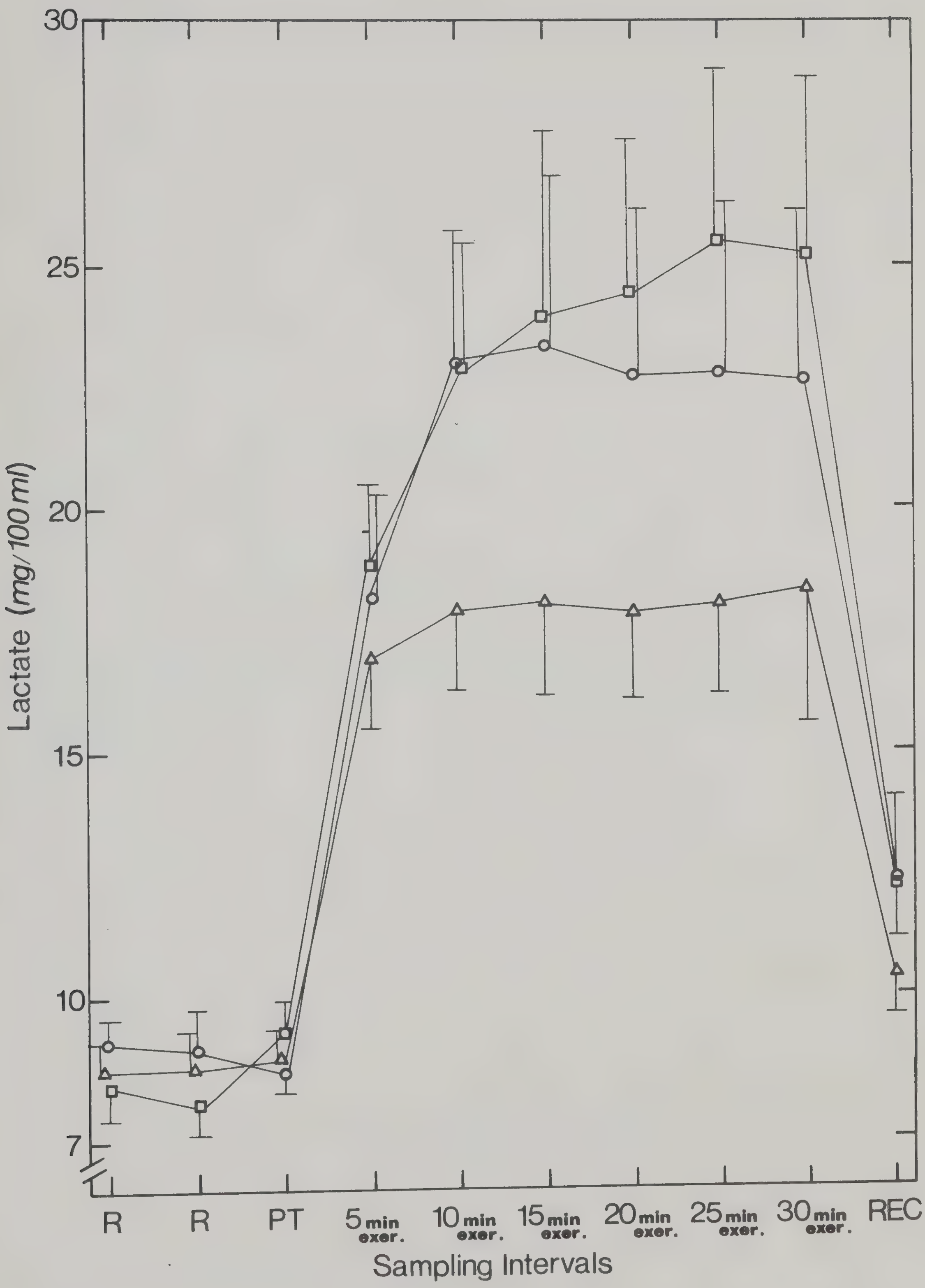
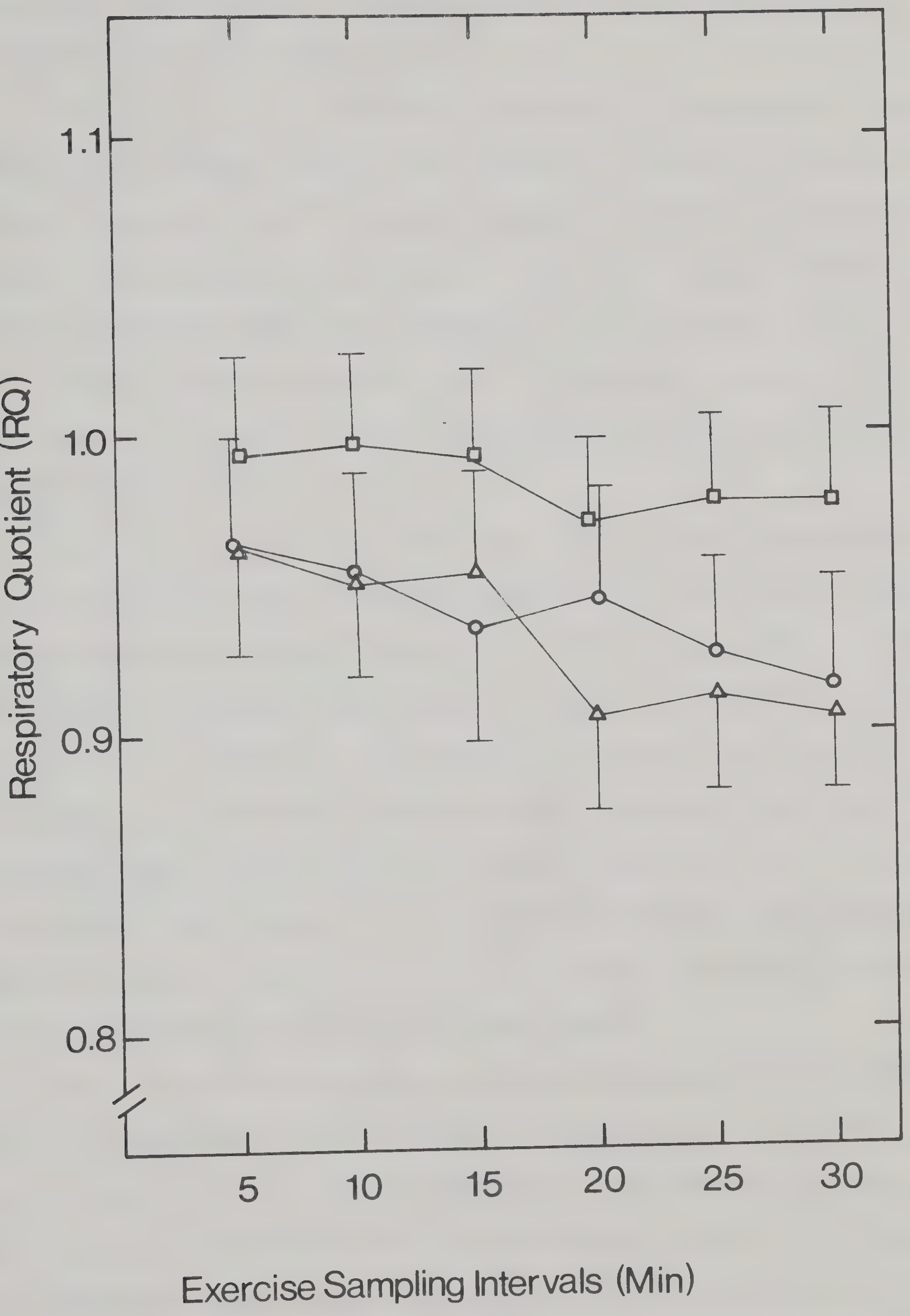

30
25
20
15
10
7
Lactate (mg/100 ml)
R
R
PT
5 min exer.
10 min exer.
15 min exer.
20 min exer.
25 min exer.
30 min exer.
REC
Sampling Intervals

FIGURE 5

Comparison of Respiratory Quotient Means ($\pm S_{\bar{e}}$) for Control (o), Exercise (▲) and Sauna (◘) Treatment Conditions During the Criterion Exercise.


1.1
1.0
0.9
0.8
Respiratory Quotient (RQ)
5
10
15
20
25
30
Exercise Sampling Intervals (Min)

insignificant decline in R.Q. from beginning to end of the 30 minutes of exercise. Exercise R.Q. in subjects warmed up in the sauna appeared somewhat higher than either T_e or T_c.

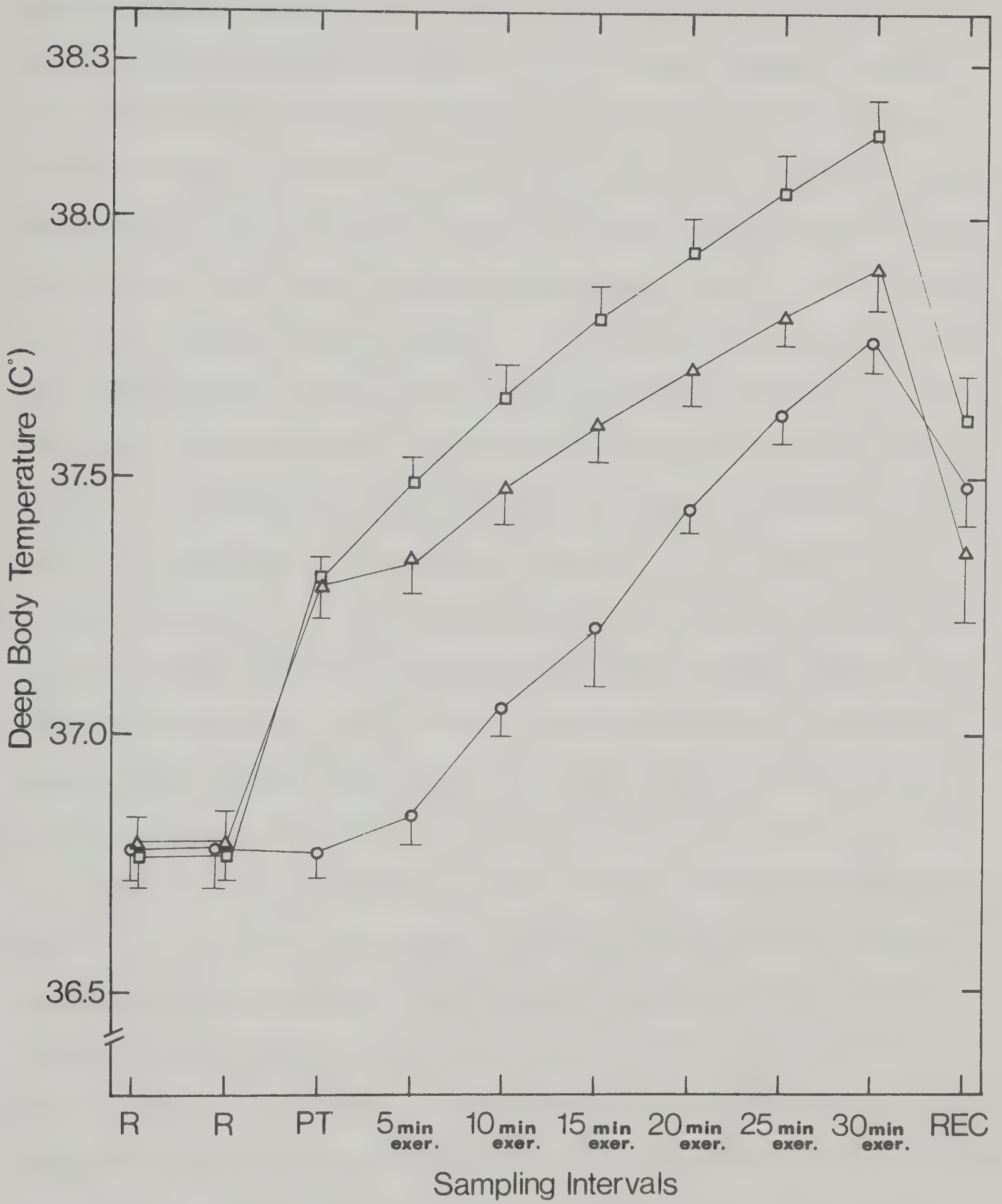
The means and $S_{\bar{e}}$ for deep body temperature at each sampling point during each treatment condition are presented in Figure 6. Both main effects (sampling times and treatment groups) and interaction effects as well showed significant differences (Table 16). One-way analysis of variance indicated significant differences for each treatment condition over all the sampling intervals; as well, significant treatment effects were shown to be present for the post treatment through to the 30 minute exercise sample (Tables 17 to 19).

Under all three treatment conditions, the deep body temperatures at the two resting samples were significantly lower than the last four samples during the exercise criterion test and recovery sample. In the two treatment conditions where warm-up was used, the deep body temperature at the two rest samples was also significantly lower than the post treatment, 5, and 10 minute exercise samples. Although in the post treatment sample, two of the three treatment conditions were 0.5°C above resting deep body temperature, the temperatures reached during the last fifteen minutes of exercise for all three treatment conditions were significantly higher than the post treatment samples.

Only for subjects who warmed up using exercise did their 15 minute recovery temperature return to the post treatment level. For both T_c and T_s, their 15 minute recovery temperature was significantly higher over their post treatment level. As can be seen in Figure 6, the deep body temperature for all three treatment conditions during exercise exhibited a continual rise with no signs of achieving a steady state.

FIGURE 6

Comparison of Deep Body Temperature Means ($\pm S_{\bar{e}}$) for Control (o), Exercise (△), and Sauna (□) Treatment Conditions During Rest, Exercise and Recovery


Deep Body Temperature (C°)
38.3
38.0
37.5
37.0
36.5
R
R
PT
5 min exer.
10 min exer.
15 min exer.
20 min exer.
25 min exer.
30 min exer.
REC
Sampling Intervals

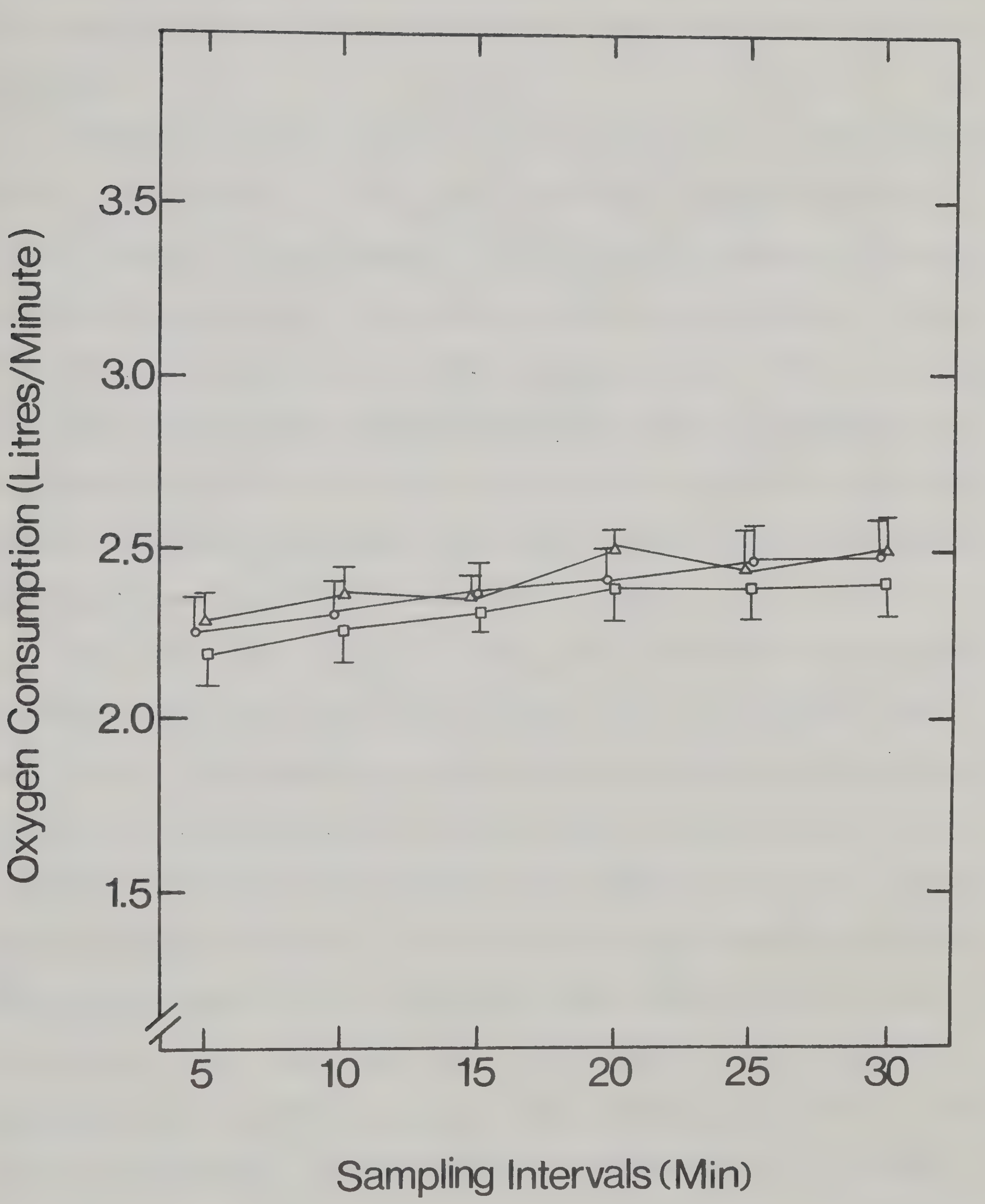
All treatment conditions exhibited significantly higher deep body temperatures: at the 20, 25, and 30 minute sampling intervals over the 5 minute sampling interval; at 25 and 30 minute sampling intervals over the 10 minute sampling interval; and the 30 minute sampling interval over the 15 minute sampling interval. In addition, the control and exercise warm-up conditions showed a significant increase in deep body temperature for the 15 minute over the 5 minute sampling interval; for the 20 minute over the 10 minute exercise sample; and for the 25 minute over the 15 minute exercise sample. For the control treatment condition, the temperatures at the 20 and 30 minute sampling intervals were significantly higher than the 15 and 20 minute sampling intervals, respectively. In recovery all three treatment conditions exhibited a fall in deep body temperature. The exercise warm-up condition exhibited the lowest temperatures of the three treatment conditions. The control and sauna treatment conditions followed with slightly higher temperatures. Recovery temperatures for all three treatment conditions were significantly lower ($p < .05$) than their respective 30 minute exercise temperature (Tables 17 to 19).

Analysis for any treatment effects showed significant differences across T_c, T_e, and T_s at the post treatment through to the 30 minute exercise sampling interval. For the post treatment, 5, 10, and 15 minute exercise samples, T was significantly lower than T_e and T_s (Tables 20, 21, 22, and 23). At the 20 minute exercise sample, all were significantly different from each other, with the sauna warm-up treatment condition being the highest followed by control and exercise warm-up treatment conditions. For the 25 and 30 minute sampling intervals, T_s was significantly higher than T_c and T_e (Tables 25 and 26).

The means and $S_{\bar{e}}$ for $\dot{V}O_2$ at each sampling point for each treatment condition are presented in Figure 7. The two-way analysis of variance showed neither 'A' nor 'B' significant ($p > .05$) main effects nor any interaction (Table 27).

FIGURE 7

Comparison of Oxygen Consumption Means (± $S_{\bar{e}}$) for Control (o), Exercise (Δ), and Sauna (□) Treatment Conditions During Rest, Exercise, and Recovery


Oxygen Consumption (Litres/Minute)
3.5
3.0
2.5
2.0
1.5
5
10
15
20
25
30
Sampling Intervals (Min)

DISCUSSION

Direct comparisons with other studies in this area are difficult to make since this study is one of the first to attempt to look at warm-up in terms of deep body temperature and its effect on plasma FFA concentration.

Existing literature (Pruett, 1970; Horstman et al., 1971; Issekutz et al., 1965a; Costill, 1970) appears to infer a critical point in aerobic capacity at approximately 70% $\dot{V}O_2$ max above which blood lactate increases significantly. Since blood lactate has been shown to inhibit FFA mobilization (Boyd et al., 1974; Issekutz et al., 1965b), a relative workload of 60% $\dot{V}O_2$ max was selected for this study in an attempt to eliminate blood lactate as a factor in altering plasma FFA levels. Fit subjects ($\dot{V}O_2$ max $\geq$ 45 ml/kg) were chosen to avoid early lactate build up usually seen in unfit subjects (Davies, 1968).

Basal FFA levels obtained in this study fall within the range observed by Frederickson and Gordon (1958), Eaton et al. (1969), and Keul (1972). The range for resting FFA concentrations for fasting subjects in this study was 0.586 to 0.641 μEq/ml. Although none of the 3 treatment conditions showed significantly different FFA levels from rest through recovery, (Figure 2), all exhibited trends reported by others (Carlson and Pernow, 1959; Carlson and Pernow, 1961; Costill et al., 1973; Basu et al., 1960; Boyd et al., 1974; Friedberg et al., 1960; Friedberg et al., 1963; Fink et al., 1975; Rodahl et al., 1964). This trend is as follows: unchanged or decreased FFA levels for the first 15 to 20 minutes followed by an increase with the largest values seen in recovery. This trend was best shown by viewing the change in FFA concentration (Δ FFA) from the initial rest samples (Figure 3). Even if

identical exercise tests are repeated (Boyd et al., 1974), FFA values need not always increase significantly from rest. Existing literature (Paul and Holmes, 1975; Pernow and Saltin, 1971) however, appears to support the concept that FFA concentration increases under submaximal exercise, despite the lack of reported statistical significance in some research (Carlson and Pernow, 1959; Carlson and Pernow, 1961; Friedberg et al., 1960; Friedberg et al., 1963; Havel et al., 1963; Havel et al., 1967; Issekutz and Miller, 1962b; Rodahl et al., 1964). Results from this experiment describing FFA concentrations are in line with results found in these previous investigations.

It appears from this study that increasing the deep body temperature 0.5°C through an active or a passive warm-up had no effect on plasma FFA levels before and during exercise at 60% $\dot{V}O_2$ max. However, the exercise warm-up condition did show the largest FFA values for the 15 minutes of exercise and recovery (Figure 2), as well as the largest increases for the last 2 exercise and recovery samples (Figure 3). The significant ($p < .05$) effect seen for exercise warm-up over control treatment condition in recovery may be due to the extended period of exercise carried out for the exercise warm-up condition. Research on FFA increases during recovery is conflicting and provides little meaningful information as to the amount of increase or as to the time at which the greatest increase occurs (Rodahl et al., 1964; Johnson et al., 1969; Cobb and Johnson, 1963; Issekutz et al., 1965a).

Lack of FFA change with either sauna or exercise warm-up to an increase of 0.5°C in deep body temperature in this study compares favorably with Britton (1974) and Taggert (1972) who found little FFA increases with heat exposures, and with Horstman et al. (1971) who

found a slight decrease in FFA with exercise warm-up of 40 to 50% $\dot{V}O_2$ max. Sauna exposure time used by Britton et al. (1974) and Taggert et al. (1972) was 10 or 15 minutes and is similar to the time spent in the sauna by subjects in this study (15.6 ± 1.12 minutes). Although Eddy et al. (1976) reported significant increases of FFA levels in subjects who dehydrated in a climatic chamber over subjects who were also in a climatic chamber but were allowed to replenish fluids, their (Eddy et al., 1976) procedure differed in many respects from that used in the present study. This may explain why FFA changes were observed by Eddy et al. (1976) and not found in this study, by Britton et al. (1974), or by Taggert et al. (1972).

The duration of the exercise needed to increase deep body temperature 0.5°C was 22.8 ± 1.47 minutes; slightly greater than the 15 minutes of exercise warm-up arbitrarily set by Horstman et al. (1971) for his subjects. This 15 minute exercise duration falls within the period that FFA levels drop below resting values and therefore may explain why FFA values for these subjects (Horstman et al., 1971) had decreased over rest. In the present study, the duration of warm-up, may have been the reason why FFA values were slightly elevated for this post warm-up sample.

Although measurement of plasma FFA concentration gives no indication as to FFA turnover, there is some evidence that turnover is highly related to concentration (Armstrong et al., 1961; Haggenfeldt and Wahren, 1975a and b). A high plasma FFA concentration therefore promotes a greater use of fat by the body. In all three treatment conditions, there was an increase in FFA concentration during exercise as well as a small, insignificant decrease in R.Q. This evidence appears to imply an

increase in fat utilization by the subjects of this study under all treatment conditions. Paul and Holmes (1975, p. 176) point out:

> There is an inverse relationship between the amount of glycogen stored inside the muscle, its rate of depletion, and muscular endurance during prolonged strenuous work. Oxidation of FFA spares muscle glycogen and in this way increases work endurance.

The concept of glycogen-sparing by FFA would therefore be in line with the results found in the present study.

A rise in deep body temperature of 0.5°C, whether achieved actively or passively, was not a significant factor in altering FFA concentrations during the half-hour exercise test at any given sampling interval.

Resting lactate values for control, exercise, and sauna treatment conditions fell in the range of 7 to 9 mg%. These values are in agreement with resting lactates reported by Costill (1970), Rennie and Johnson (1974), Fink et al. (1975), and Eddy et al. (1976). Neither sauna nor exercise warm-up appeared to alter the lactate values (Figure 4) from rest. This lack of change of lactate in the sauna cannot be explained due to a paucity of related literature. As the heat stress was only passive in nature, it would appear that being in the heat does not differ from being in normal environmental temperature with respect to lactate production. Lactate response for subjects immediately following the active warm-up also did not change. This was expected, though, based on the finding of Pruett (1970) who observed little or no increase in lactate for subjects working below 50% $\dot{V}O_2$ max.

It appears from this study that if exercise is used as a warm-up rather than the sauna or no warm-up at all, lower lactates result (Figure 4) when, subsequently, subjects exercise at a higher intensity, 60% $\dot{V}O_2$ max. This finding is in agreement with that observed by Martin

(1975) who found lower lactates for all exercise tests preceeded by an exercise warm-up.

Lactate levels reported by Pruett (1970) for subjects working at 70% $\dot{V}O_2$ max were 20 to 25 mg%. These values did not interfere with increases in FFA concentrations for her (Pruett, 1970) subjects, and compared favorably with lactate values obtained in this study. Exercise lactate values obtained at 60% $\dot{V}O_2$ max in this study ranged from 25.5 mg% for the sauna warm-up condition to 23.4 and 18.4 mg% for the control and exercise warm-up conditions respectively. For subjects who received exercise as a warm-up, their final 30 minute lactate sample was the only one to increase significantly ($p < 0.05$) over rest, while all exercise lactates in the other two treatment conditions were significantly ($p < 0.05$) greater than resting values. However, this low quantity of lactate, whether significantly increased from rest or not, must increase to between 60 to 70 mg% (Issekutz et al., 1975) to be of physiological importance in the inhibition of FFA release from adipose tissue. Lactate values reported by Claremont et al. (1975) were somewhat higher (35.9 and 26.5 mg%) for exercise at 52 to 59% $\dot{V}O_2$ max. His (Claremont et al., 1975) subjects, however, exercised at 2 different ambient temperatures of 35° and 0°C. Claremont et al. (1975, p. 151) suggested:

> ...the higher lactate at 35°C is probably due to decreased lactic acid uptake by the liver, not tissue hypoxia.

But this higher value could be explained by differences in the fitness level of the subjects in the two studies. Higher circulating lactate levels were reported as well by Rowell (1965) in conjunction with a decrease in liver blood flow when subjects exercised at an elevated environmental temperature. In this study, subjects only warmed up

passively in the sauna, and the subsequent exercise test was conducted in a comfortable laboratory environment (22°C). The lactate levels during this exercise test, though, were the highest values elicited in any of the three treatment conditions. Possibly the decrease in liver blood flow reported by Rowell et al. (1965) may have been a factor during resting sauna conditions, followed by exercise at 22°C. Measurement of increased lactate production as well as increased glucose utilization for subjects exercised in the heat prompted Fink et al. (1975, p. 188) to state:

> Although Rowell et al. ... have shown reduced rate of hepatic lactate removal during work in heat, the greater blood lactate values observed in the present study are also the result of an increased rate of anaerobic metabolism.

In this present study, subjects who warmed up using the sauna exhibited the following; the highest R.Q. (Figure 5); FFA below resting levels for 25 of the 30 minutes of exercise (Figure 3); and the highest lactate response for the entire exercise period (Figure 4). Considering the fitness of the subjects and the 60% $\dot{V}O_2$ max work load, it would be safe to assume that aerobic metabolism was occurring for this particular treatment condition. Both Nagle et al. (1970) and Costill (1970) have noted that, in fit subjects, lactate elevation through anaerobic metabolism increases in proportion to aerobic demands in excess of 65 to 70% $\dot{V}O_2$ max. In light of this present study, it appears that exercise as a warm-up reduces the lactate response in subsequent exercise, while sauna warm-up seems to produce the opposite effect. Although FFA concentrations exhibited an increase for all three conditions, these increases were not significant ($p > 0.05$) from rest. However, in recovery the exercise warm-up condition produced FFA values significantly ($p < 0.05$)

elevated above the control exercise sample only.

Hence, it seems that the lower lactate levels during the 60% $\dot{V}O_2$ max test did not alter the FFA levels during the exercise bout. Although it is not possible to imply FFA turnover rates from blood levels alone, the R.Q. data would suggest no significant alteration in metabolic fuel as a result of any of the treatment conditions.

There was no significant change ($p > 0.736$) in R.Q. from beginning to end of the 30 minutes of exercise (Table 14). All three treatment conditions showed insignificant decreases in R.Q. as work progressed (Figure 5), indicative of a slow shift toward fat metabolism. These decreases were mirrored by the small increases in FFA concentration for all three treatment conditions as exercise progressed. Miller et al. (1963) points out that there are inherent problems in interpreting this ratio (R.Q.). He (Miller, et al., 1963; p. 167) states:

> During work, lactic acid and other acids will be formed and therefore a certain portion of expired CO_2 is derived from the bicarbonate pool and not from the oxidation of any fuel. This excess CO_2 elevates the $CO_2:O_2$ ratio and may lead to the conclusion that the metabolism during work has shifted toward carbohydrate oxidation.

Although interaction between treatments was indicated initially by the two-way analysis of variance, subsequent one-way analysis showed no significant ($p > 0.05$) interaction between any of the three treatments at any of the sampling points. Exercise RQ following sauna warm-up tended to be slightly elevated over control and exercise warm-up conditions. This perhaps can be partly explained by the elevated lactate levels seen in the exercise following sauna warm-up or by a slight shift towards carbohydrate as the metabolic substrate.

The half hour of exercise was insufficient time to observe a

plateau in deep body temperature for any of the 3 treatment conditions (Figure 6). This continual rise of deep body temperature was responsible for a significant ($p < 0.001$) 'A' main effect (Tables 16 to 18) for each treatment condition. Although Saltin (1966) suggests that a steady state may be reached within 20 minutes, there was no sign of this in the present study. The conflicting factor appears to be that, while the external temperature was kept the same (22°C), the starting deep body temperature was raised through warm-up for 2 of the 3 treatment conditions. Significant interaction ($p < 0.05$) between treatment conditions resulted at every sampling point during the 30 minutes of exercise (Tables 19 to 25). Temperatures for the sauna warm-up condition exhibited the highest levels throughout the 30 minutes of exercise and recovery and were significantly different from either the control or exercise warm-up conditions during the last 10 minutes of exercise.

There is a lack of research at this time to relate how initial differences in deep body temperature affect increases in temperature during subsequent exercise. Indirectly, one may gather some information by looking at the changes in deep body temperature as a result of exercise conducted at various ambient temperatures. The effects of environmental temperature on work performance show that increases in deep body temperature are similar for at least the first 20 to 30 minutes of exercise (Fink et al., 1975; Claremont et al., 1975). At the end of 30 minutes of exercise at 52 to 59% VO_2 max and 0° and 35°C, subjects in both temperature conditions had a similar increase of 0.7°C in deep body temperature (Claremont et al., 1975). This compares favorably with increases found in this study (0.988°C for control; 0.872°C for sauna warm-up; and 0.716°C for the exercise warm-up

treatment conditions). Fink et al. (1975) exercised subjects at 9° and 41°C and observed that, while working at 70 to 80% $\dot{V}O_2$ max, the rise in deep body temperature was similar (0.7 to 0.8°C) for both conditions for at least 20 to 25 minutes.

Oxygen consumption did not change significantly ($p > 0.216$) throughout the 30 minutes of exercise for any of the three treatment conditions (Table 27). In addition, the warm-up effect of exercise and sauna produced no significant interaction ($p > 0.16$) over the three treatment conditions. Oxygen uptake increased slightly toward the end of the 30 minutes of exercise, reaching a mean value for all three treatment conditions of 2.44 ± 0.064 liters of oxygen per minute (Figure 7). The mean workload at which this was achieved was 1002.3 ± 25.33 kpm/minute. The oxygen consumption compares favorably with the value of 2.4 liters obtained using the Astrand-Rhyming nomogram (Astrand, 1960). Holloszy (1975, p. 160) states:

> ...oxygen consumption is the same in the trained and untrained state during submaximal work of a given intensity.

Based on this study, one may add as well that changes in deep body temperature (a rise of 0.5°C), achieved either via exercise or sauna warm-up prior to a submaximal workload of a given intensity, will elicit the same oxygen consumption for the submaximal workload.

In light of the fact that $\dot{V}O_2$ max and weight of the subjects in this study did not change from beginning to end, it eliminates the possibility of any interference occurring due to a training or detraining effect.

Summary

For two of the three experimental treatment conditions, deep body

temperature was elevated 0.5°C in an active (T_e: exercise at 40% $\dot{V}O_2$ max) or passive (T_s: sitting in a sauna) manner. The third treatment condition served as control (T_c: no elevation in deep body temperature). Each treatment condition was followed by a 30-minute exercise at 60% $\dot{V}O_2$ max (CT).

Free fatty acids did not change significantly from rest for any of the treatment conditions, although all three displayed the similar trend of an increased FFA level toward the end of the 30 minutes of exercise. Warm-up of 0.5°C in deep body temperature did not immediately alter the plasma concentration of FFA for the two experimental groups (T_s and T_e), nor did it significantly affect the FFA concentration throughout the subsequent 30 minutes of exercise. Subjects who performed T_e exhibited significantly higher FFA levels in recovery than T_c.

Plasma lactate was not altered from rest by either of the two warm-up conditions. With the subsequent 30 minutes of exercise, all three treatment conditions increased plasma lactate significantly. Subjects who experienced exercise as a warm-up (T_e) had significantly lower lactates than either T_c or T_s for the final 25 of the 30 minutes of exercise.

All conditions exhibited a significant rise in deep body temperature during the 30 minutes of exercise. There was no sign of a plateau for any of the three treatment conditions. Sauna warm-up (T_s) elicited the highest temperatures at each sampling point during the 30 minutes of exercise, followed by T_e and T_c. T_s showed significantly higher temperatures than either T_c or T_e at the 25 and 30 minute exercise samples.

Warm-up of 0.5°C in deep body temperature had no significant effect on exercise RQ nor on oxygen consumption. Subjects who had sauna warm-up exhibited slightly greater RQ values. RQ for all three treatment

conditions tended to decline as exercise progressed, while oxygen consumption tended to show a slight increase.

CONCLUSIONS

Within the limits of this study, the following conclusions may be drawn:

1. Warm-up via exercise at approximately 40% $\dot{V}O_2$ max and sauna exposure as indicated by an increase in deep body temperature of 0.5°C does not alter FFA concentrations over resting values;

2. Exercise of short duration (30 minutes at 60% $\dot{V}O_2$ max) is insufficient in either duration, intensity or both to demonstrate any changes in FFA concentration, regardless of the type of preliminary warm-up;

3. Lactate levels during exercise of submaximal intensity (60% $\dot{V}O_2$ max) are significantly less when preceded by an exercise warm-up than those found after a passive warm-up or no warm-up at all;

4. Increases in deep body temperature during exercise seem to be significantly increased when preceded by a sauna warm-up rather than an exercise warm-up or no warm-up at all;

5. Although blood lactate levels during exercise preceded by exercise warm-up would imply reduced use of carbohydrate as a fuel, RQ values did not support this. R.Q. did not give any positive indication as to whether warm-up altered the type of fuel utilized during subsequent exercise;

6. Oxygen consumption during exercise of submaximal intensity is not affected by a previous warm-up of 0.5°C in deep body temperature.

REFERENCES

REFERENCES

Andres, R., Cader, G., and Zierler, K. The quantitatively minor role of carbohydrate in oxidative metabolism by skeletal muscle in the basal state. Measurements of O_2 and glucose uptake in the forearm. J. Clin. Invest. 35: 671-682, 1956.

Armstrong, O., Steele, R., Altszuler, N., Dunn, A., Bishop, J. and DeBodo, R. Regulation of plasma free fatty acid turnover. Am. J. Physiol. 201: 9-15, 1961.

Asmussen, E. and Boje, O. Body temperature and capacity for work. Acta Physiol. Scand. 10: 1-22, 1945.

Astrand, I. Aerobic work capacity in men and women with special reference to age. J. Appl. Physiol. 49: suppl. 169, 1960.

Astrand, P. Hallback, I., Hedman, R. and Saltin, B. Blood lactates after prolonged severe exercise. J. Appl. Physiol. 18: 619-622, 1963.

Astrand, P. and Rodahl, K. Textbook of Work Physiology. Toronto: McGraw-Hill Publishers, 1970.

Barcroft, J. and King, W. The effect of temperature on the dissociation curve of blood. J. Physiol. Lond. 39: 374-384, 1910-11.

Basu, A., Passmore, R. and Strong, J. The effect of exercise on the level of non-esterified fatty acids in the blood. Quart. J. Exp. Physiol. 45: 312-317, 1960.

Bell, C., Hellon, R., Hiorns, R., Nicol, P. and Provins, K. Safe exposure of men to severe heat. J. Appl. Physiol. 20: 288-292, 1965.

Bodel, P., Rubenstein, D., McGarry, E. and Beck, J. Utilization of free fatty acids by diaphragm in vitro. Am. J. Physiol. 203: 311-315, 1962.

Boyd, A., Giamber, S., Mager, M. and Lebovitz, H. Lactate inhibition of lipolysis in exercising man. Metabolism 23: 531-542, 1974.

Britton, B., Hawkey, C., Wood, W., Peele, M. and Irving, M. Adrenergic coagulation and fibrinolytic responses to heat. Bri. Med. J. 4: 139-141, 1974.

Bruce, R., Cobb, L. and Williams, R. Effect of exercise and isoproterenol on free fatty acids and carbohydrates in cardiac patients. Am. J. Med. Sci. 241: 59-67, 1961.

Carlson, L. and Pernow, B. Studies on blood lipids during exercise. I. Arterial and venous plasma concentrations of unesterified fatty acids. J. Lab. Clin. Med. 53: 833-841, 1959.

Carlesten, A., Gustafson, A. and Werko, L. Hemodynamic influence of warm and dry environment in man with and without rheumatic heart disease. Acta Med. Scand. 169: 411-417, 1961.

Carlson, L. and Pernow, B. Studies on blood lipids during exercise. II. The arterial plasma free fatty acid concentration during and after exercise and its regulation. J. Lab. Clin. Med. 58: 673-681, 1961.

Carlson, L., Boberg, J. and Hogstedt, B. Adipose tissue. Handbook of Physiology. Edited by A. Renold and G. Cahill. Washington, D.C.: American Physiological Society, Chapter.63, p. 625, 1965.

Claremont, A., Nagle, F., Reddan, W. and Brooks, G. Comparison of metabolic temperature, heart rate, and ventilatory responses to exercise at extreme ambient temperatures (0° and 35°C). Med. Sci. Sports. 7: 150-154, 1975.

Clark, A., Gaddie, R. and Stewart, C. The aerobic metabolism of the isolated frogs heart poisoned by iodoacetic acid. J. Physiol. (London) 90: 335-346, 1937.

Cobb, L. and Johnson, W. Hemodynamic relationship of aerobic metabolism and plasma free fatty acids during prolonged, strenuous exercise in trained and untrained subjects. J. Clin. Invest. 42: 800-810, 1963.

Costill, D. Metabolic responses during distance running. J. Appl. Physiol. 28: 251-255, 1970.

Costill, D., Thomason, H. and Roberts, E. Fractional utilization of aerobic capacity during distance running. Med. Sci. Sports, 5: 248-252, 1973.

Christensen, E. and Hansen, O. Arbeitsfahigkeit und Ehrnahrung. Skand. Arch. Physiol. 81: 160-169, 1939.

Cumming, G. and Borysyk, L. The maximal exercise E.C.G. in asymptomatic men. Can. Med. Ass. J. 106-110, 1972.

Davies, C. and Harris, E. Heart rate during transition from rest to exercise in relation to exercise tolerance. J. Appl. Physiol. 19: 857-862, 1964.

Davies, C. Cardiac frequency in relation to aerobic capacity for work. Ergonomics 11: 511-526, 1968.

DeVries, H., Beckman, P., Huber, H. and Dieckmeir, L. Electromygraphic evaluation of the effects of sauna on the neuromuscular system. J. Sports Med. 8: 61-69, 1968.

Dole, V. A relation between non-esterified fatty acids in plasma and the metabolism of glucose. J. Clin. Invest. 35: 150-154, 1956.

Eaton, R. and Steinberg, O. Effects of medium fatty acid concentration, epinephrine and glucose on palmitate $1^{-14}C$ oxidation and incorporation in neutral lipids by skeletal muscle in vitro. J. Lip. Res. 2: 376-382, 1961.

Eaton, R., Bearman, M. and Steinberg, D. Kinetic studies of plasma FFA and triglyceride metabolism in man. J. Clin. Invest. 40: 1560-1579, 1969.

Eddy, C., Sparks, K. and Turner, C. The adipokinetic effect of hyperthermic stress in man. Eur. J. Appl. Physiol. 35: 103-110, 1976.

Eisalo, A. Effects of a Finnish Sauna on circulation. Annals Med. Exp. Biol. Finn. 34(suppl. 4): 1-96, 1956.

Fink, W., Costill, D. and Vanhandel, P. Leg muscle metabolism during exercise in the heat and cold. Eur. J. Appl. Physiol. 34: 183-190, 1975.

French, J., Morris, B. and Robinson, D. Removal of lipids from the blood stream. Bri. Med. Bull. 14: 234-238, 1958.

Fredrickson, D. and Gordon, R. The metabolism of albumin-bound C^{14}-labeled unesterified fatty acids in normal human subjects. J. Clin. Invest. 37: 1504-1515, 1958.

Friedberg, S., Harlan, W., Trout, D. and Estes, E. The effects of exercise on the concentration and turnover of plasma nonesterified fatty acids. J. Clin. Invest. 39: 215-221, 1960.

Friedberg, S., Sher, P., Bogdonoff, D. and Estes, E. The dynamics of plasma free fatty acid metabolism during exercise. J. Lip.Res. 4: 34-38, 1963.

Fritz, I. Factors influencing the rates of long-chain fatty acid oxidation and synthesis in mammalian systems. Physiol. Rev. 41: 52-129, 1961.

Gemmill, C. The fuel for muscular exercise. Physiol.Rev. 22: 32-53, 1942.

Goldman, R., Green, E. and Impietro, P. Tolerance of hot, wet environments by resting men. J. Appl. Physiol. 20: 271-277, 1965.

Gordon, R. and Cherkes, A. Unesterified fatty acid in human blood plasma. J. Clin. Invest. 35: 206-212, 1956.

Grimby, G., Haggendahl, E. and Sältin, B. Local Xenon 133 clearance from the quadriceps muscle during exercise in man. J. Appl. Physiol. 22: 305-310, 1967.

Haggenfeldt, L. and Wahren, J. Metabolism of free fatty acids and ketone bodies in skeletal muscle. Muscle Metabolism. Edited by B. Pernow and B. Saltin, New York - London: Plenum Press, 1971.

Haggenfeldt, L. and Wahren, J. Turnover of plasma free anachidonic and oleic acids in resting and exercising human subjects. Metabolism 24: 799-806, 1975a.

Haggenfeldt, L. and Wahren, J. Turnover of free fatty acids during recovery from exercise. J. Appl. Physiol. 39: 247-250, 1975b.

Havel, R., Naimark, A. and Borchgrevink, C. Turnover rate and oxidation of free fatty acids of blood plasma in man during exercise: Studies during continuous infusion of palmitate-1-C^{14}. J. Clin. Invest. 42: 1054-1062, 1963.

Havel, R., Pernow, B. and Jones, N. Uptake and release of free fatty acids and other metabolites in the legs of exercising man. J. Appl. Physiol. 23: 90-99, 1967.

Havel, R. Influence of intensity and duration of exercise on supply and use of fuels. Muscle Metabolism During Exercise. Edited by B. Pernow and B. Saltin, New York-London: Plenum Press, 1971.

Hermansen, L., Hultman, E. and Saltin, B. Muscle glycogen during prolonged severe exercise. Acta Physiol. Scand. 71: 129-131, 1967.

Holloszy, J. Adaptations of skeletal muscle to endurance exercise. Med. Sci. Sports 7: 155-164, 1975.

Horstman, D., Mendez, J., Buskirk, E., Boileau, R. and Nicholas, W. Lipid metabolism during heavy and moderate exercise. Med. Sci. Sports 3: 18-23, 1971.

Howald, H. and Poortmans, J. Metabolic Adaptations to Prolonged Physical Exercise. Switzerland: Birkhauser Verlag Basel, 1975.

Issekutz, B. and Miller, H. Plasma free fatty acids during exercise and the effect of lactic acid. Proc. Soc. Exp. Bio. Med. 110: 237-239, 1962.

Issekutz, B., Birkhead, N. and Rodahl, K. Effect of diet in work metabolism. J. Nut. 79: 109-115, 1963.

Issekutz, B., Miller, H., Paul, P., Rodahl, K. Source of fat oxidation in exercising dogs. Am. J. Physiol. 207: 583-589, 1964.

Issekutz, B., Miller, H., Paul, P. and Rodahl, K. Aerobic work capacity and plasma FFA turnover. J. Appl. Physiol. 20: 293-296, 1965a.

Issekutz, B., Miller, H., Paul, P. and Rodahl, K. Effect of lactic acid on free fatty acids and glucose oxidation in dogs. Am. J. Physiol. 209: 1137-1144, 1965b.

Issekutz, B., Miller, H. and Rodahl, K. Lipid and carbohydrate metabolism during exercise. Fed. Proc. 25: 1415-1420, 1966.

Issekutz, B., Shaw, W. and Issekutz, T. Effect of lactate on FFA and glycerol turnover in resting and exercising dogs. J. Appl. Physiol. 39: 349-353, 1975.

Johnson, R., Walten, J., Drebs, H. and Williamson, D. Metabolic fuels during and after severe exercise in athletes and non-athletes. Lancet, 2: 452-455, 1969.

Karlsson, J. Lactate in working muscles after prolonged exercise. Acta Physiol. Scand. 82: 123-130, 1971.

Keul, J., Doll, E. and Keppler, D. Energy metabolism in human muscle. Medicine and Sport. Edited by E. Jokl, Baltimore: University Park Press, 1972.

Keul, J., Haralambie, G., Arnold, T. and Schumann, W. Heart rate and energy-yielding substrates in blood during long-lasting running. Eur. J. Appl. Physiol. 32: 279-289, 1974.

Lind, A. A physiological criterion for setting thermal environmental limits for every day work. J. Appl. Physiol. 18: 51-56, 1963.

Lehninger, A. The oxidation of higher fatty acids in heart muscle suspensions. J. Biol. Chem. 165: 133-145, 1946.

Martin, B., Robinson, S., Wiegman, D. and Aulick, L. Effect of warm-up on metabolic responses to strenuous exercise. Med. Sci. Sports 7: 124-149, 1975.

Miller, H., Issekutz, B. and Rodahl, K. Effects of exercise on the oxidation of fatty acids in the dog. Am. J. Physiol. 205: 167-172, 1963.

Nagle, F., Robinhold, D., Howley, F., Daniels, J., Batista, C. and Stoedefalke, K. Lactic acid accumulation during running at submaximal aerobic demands. Med. Sci. Sports 2: 182-186, 1970.

Newsholme, E. and Start, C. Regulation in Metabolism. London: John Wiley and Sons, 1973.

Nielsen, M. Die regulation der korpertemperature bie muskelarbeit. 1938. Quoted from P. Astrand and K. Rodahl, Textbook of Work Physiology. Toronto: McGraw-Hill, 1970.

Nielsen, B. and Nielsen, M. Body temperature during work. Acta Physiol. Scand. 56: 120-129, 1962.

Owen, O. and Reichard, G. Fuels consumed by man: the interplay between carbohydrates and fatty acids. Progress in Biochemical Pharmacology. Edited by R. Padletti, Switzerland: Karger Basal, 1971.

Paul, P. and Issekutz, B. Role of extramuscular energy sources in the metabolism of exercising dog. J. Appl. Physiol. 22: 615-622, 1967.

Paul, P. FFA metabolism of normal dogs during steady state exercise at different workloads. J. Appl. Physiol. 28: 127-130, 1970.

Paul, P. and Holmes, W. Free fatty acid and glucose metabolism during increased energy expenditure and after training. Med. Sci. Sports 7: 176-184, 1975b.

Pernow, P. and Saltin, B. (Editors) Muscle Metabolism During Exercise. New York: Plenum Press, 1971.

Pinelli, A. A new colorimetric method for plasma fatty acid analysis. Clin. Chim. Acta 44: 385-390, 1973.

Pugh, L. Cold stress and muscular exercise with special reference to accidental hypothermia. Bri. Med. J. 2: 333-337, 1967.

Pruett, E. FFA mobilization during and after prolonged severe muscular work in men. J. Appl. Physiol. 29: 809-815, 1970.

Rennie, M. and Johnson. R. Effects of an exercise diet program on metabolic changes with exercise in runners. J. Appl. Physiol. 37: 821-825, 1974.

Robinson, S., Meyer, F., Newton, J., Tsas, C. and Holgersen, L. Relations between sweating, cutaneous blood flow and body temperature in work. J. Appl. Physiol. 20: 575-582, 1965.

Rodahl, K., Miller, H. and Issekutz, B. Plasma free fatty acids in exercise. J. Appl. Physiol. 19: 489-492, 1964.

Rowell, L., Blackman, J., Martin, R., Mazzarella, J. and Bruce, R. Hepatic clearance of indocyanine dye in man under thermal exercise stresses. J. Appl. Physiol. 20: 384-394, 1965.

Saiki, H., Margaria, R. and Cuttica, F. Lactic acid production in submaximal work. Int. Z. Angew. Physiol. Arbeitsphysiol. 24: 57-61, 1967.

Saltin, B. and Hermansen, L. Esophageal, rectal and muscle temperature during exercise. J. Appl. Physiol. 21: 1757-1762, 1966.

Saltin, B. Body temperature and sweating during severe exercise. Environment Effects on Work Performance. Edited by G. Cumming

et al., Edmonton: University of Alberta Printing Services, 1972.

Saltin, B. Metabolic fundamentals in exercise. Med. Sci. Sports 5: 137-142, 1973.

Sancetta, S., Kramer, J. and Husni, E. The effect of dry heat on the circulation of man: general hemodynamics. Am. Heart J. 56: 212-221, 1958.

Sancetta, S., Hackel, D., Tracks, E. and Wittels, B. The effect of dry heat on the circulation of man: coronary hemodynamics. Am. Heart J. 67: 593-598, 1964.

Sigma Chemical Company. Pyruvic acid and lactic acid at 340 u. Technical Bulletin No. 726-uv and No. 826-uv. Sigma Chemical Company, 350 de Kalb Street, St. Louis, 1968.

Spector, A. Metabolism of free fatty acids. Progress in Biochemical Pharmacology. Edited by R. Paoletti, Switzerland, Karger Basel, 1971.

Stuart, D. Effects of preliminary exercise on performance. Unpublished Masters Thesis. University of Alberta, Edmonton, 1972.

Taggert, P., Parkinson, P. and Carruthers, M. Cardiac responses to thermal, physical and emotional stress. Bri. Med. J. 3: 71-76, 1972.

Taylor, A., Schoeman, D., Lovlin, R. and Lee, S. Plasma free fatty acid mobilization with graded exercise. J. Sports Med. Phys. Fit. 11: 234-240, 1971.

Wasserman, K., Burton, G. and Van Kesal, A. The excess lactate concept and the oxygen debt of exercise. J. Appl. Physiol. 20: 1299-1306, 1965.

Wasserman, K., Van Kessel, A. and Burton, G. Interaction of physiological mechanisms during exercise. J. Appl. Physiol. 22: 71-85, 1967.

Wenger, H. and Reed, A. Metabolic factors associated with muscular fatigue during aerobic and anaerobic work. Can. J. Appl. Sport Sci. 1(1): 43-48, 1976.

Weinhouse, S., Millingion, R. and Volk, M. Oxidation of isotopic palmitic acid in animal tissues. J. Biol. Chem. 185: 191-200, 1950.

Wertheimer, E. and Ben Tor, V. Fat utilization by muscle. Biochem. J. 50: 573-576, 1952.

Winer, B. Statistical Principles in Experimental Design. New York: McGraw-Hill, 1971.

APPENDIX A

APPENDIX A

REVIEW OF LITERATURE

The importance of carbohydrate and fat in energy production is well established (Howald and Poortmans, 1975). Although early research demonstrated the existence of energy production associated with the catabolism of glucose, there was no experimental proof of direct utilization of fat by skeletal muscle (Gemmill, 1942), whereas in cardiac muscle, fat catabolism appeared certain (Clark, 1937). The direct oxidation of FFA in skeletal muscle, however, was demonstrated by Lehninger (1946) through the determination of the uptake and degradation of ^{14}C-labelled fatty acids. Weinhouse et al. (1950) and Wertheimer and Ben Tor (1956) showed that muscle contained the necessary enzymes for the oxidation of lipids.

Free Fatty Acids

Free Fatty Acids (FFA) are one of three classes of lipids present in the plasma. The other two are chylomicrons, a dietary fatty acid absorbed from the gastro-intestinal tract, and lipoproteins, a complex union of lipids with protein which aid in plasma lipid solubility. The fatty acids which are important for energy supply of skeletal muscle, i.e., the triglycerides and the non-esterified fatty acids, are present in varying concentrations in the blood. The majority of lipids in blood plasma are bound to globulin, while FFA are bound to albumin (Keul et al., 1972). FFA are continuously mobilized from adipose tissue and metabolized by β-oxidation in skeletal muscle.

Values for plasma FFA in relation to the total plasma lipids vary from 2-3% (Carlson et al., 1965) to 5-10% (Spector, 1971). Plasma concentrations of FFA in normal human subjects have been observed to vary

between 0.09-1.71 mEq/1 (Friedrickson and Gordon, 1958) with the majority of cases between 0.4-0.7 mEq/1 (Eaton et al., 1969). Keul et al. (1971, p. 157) states:

> ...normal values for resting fasting man, of one who has eaten only a small amount of fat, should be between 0.3 and 0.9 uEq/ml.

Storage of lipids provides the most expedient form of energy conservation. Each molecule of fat contains approximately 9 kcal of energy per gram, compared with 4 kcal/gram for carbohydrate which is stored with water, thus reducing its efficiency as a stored fuel (Astrand and Rodahl, 1970). Plasma FFA originates primarily in the fat tissue throughout the body (Keul et al., 1971) and, to a lesser extent, FFA can be supplied by the liver through conversion of glucose or glycogen to FA or through lipolysis of triglycerides (French et al., 1958). Since plasma FFA concentration was found to be low, its importance as a fuel was questioned. Only after the work of Gordon and Cherekes (1956) and of Dole (1956) was it shown that, in spite of its small pool size and concentration, FFA have a rapid turnover rate. Intravenous infusion of palmitate-1-^{14}C in tracer amounts indicated that the half-life of plasma FFA in humans varies between 1.0 and 3.9 minutes (Eaton, 1969; Friedrickson and Gordon, 1958). When the FFA concentration is 1 uEq/ml or more, approximately 25% of the total pool leaves the plasma each minute (Friedrickson and Gordon, 1958). Carlson (1967, p. 18) points out that:

> In man, the half life time is only a few minutes and one-third to one-quarter of the plasma pool is turned over each minute.

Both in vitro and in vivo studies have shown that FFA, once delivered to the cell membranes, are readily taken up and oxidized at rest

(Friedrickson and Gordon, 1958; Bodel, 1962) and during exercise of short or long duration (Carlson and Pernow, 1959; Friedberg et al., 1963; Paul and Holmes, 1975).

Evaluation of FFA metabolism in skeletal muscle at rest and during exercise has been rendered difficult by the finding of simultaneous entry and removal of FFA from the plasma (Haggenfeldt, 1971). Armstrong et al. (1961) observed the relationship between FFA concentration and FFA uptake, noting that mobilization at an unchanging FFA concentration must equal uptake. Using adult normal dogs, variations in plasma FFA levels ranging from 0.081 to 3.31 uEq/ml were induced by a variety of physiological and pharmacological treatments (exercise excluded). Results indicated that:

> FFA production rate controls FFA concentration and that FFA concentration in turn controls FFA uptake.
> (Armstrong, 1961, p. 14)

Concentration was controlled solely by changing the rate of FFA production. Armstrong described this as a simple "mass action effect" (Armstrong, 1961, p. 9). Haggenfeldt (1971), in a series of human experiments performed after a 3-week total fast, investigated forearm exercise of specified duration, frequency, and intensity. He (Haggenfeldt, 1971, p. 157) noted that:

> ...muscle uptake of FFA rises linearly with inflow and saturation conditions do not arise even under the rather extreme circumstances of these studies. The results imply that the magnitude of FFA uptake is not regulated primarily by the muscle, but is determined largely by outside factors such as rate of lipolysis in adipose tissue.

Once delivered to the cell, FFA catabolism within the muscle may be regulated by the ability of the mitochondria to take up FFA. The

transport of fatty acids into the cell is dependent upon the concentration in the blood, but once inside the cell, they must be estrified with carnitine for transport into the mitochondria (Wenger and Reed, 1976; Newsholme and Start, 1973).

Exercise may be one of many factors that can change FFA levels in the blood (Keul et al., 1972). As energy expenditure increases during exercise, adipose tissue responds, and an increase in plasma FFA concentration is seen. The primary mechanism which controls lipolysis during exercise appears to be the increased concentrations of catacholamines (Newsholme and Start, 1973). Other agents which may play a role in altering the rate of lipolysis are insulin, glucagon growth hormone, thyroid-stimulating hormone, and adrenocorticotophic hormone. The direction of change in the plasma FFA level during exercise is dependent on the relative severity of the work as measured by oxygen uptake (Pruett, 1970). Plasma FFA concentrations first decrease as exercise begins due to an increased fractional turnover rate (Carlson and Pernow, 1959; Carlson and Pernow, 1961; and Friedberg et al., 1963). Carlson and Pernow (1959) exercised 6 human subjects on a bicycle ergometer in both sitting and supine positions. FFA concentration was lowered as the exercising leg extracted FFA from the plasma. A negative a-v difference indicated that some mobilization of FFA from fat stores was taking place. A subsequent study (Carlson and Pernow, 1961) showed that, in a 30-minute exercise period on a bicycle ergometer, FFA levels initially decreased for the first 10 minutes, after which they then increased. They (Carlson and Pernow, 1961) attributed the initial decrease in FFA concentration to an increase in blood flow to the exercising muscle, as well as an accelerated rate of removal

from the plasma. Based on a-v differences, Bruce et al. (1961) showed that, when subjects exercised on a bicycle ergometer in a supine position uptake of FFA increased regardless of blood flow. Both arterial and venous concentrations fell significantly during the four minutes of exercise.

If the exercise is aerobic in nature, demanding less than 70% of the subject's $\dot{V}O_2$ max (Pruett, 1970) FFA concentration begins to increase progressively after a duration of about 15 minutes (Basu et al., 1960; Carlson and Pernow, 1961; Friedberg et al., 1963; Havel et al., 1963). This has been attributed to increased mobilization with the highest levels reached after exercise (Carlson et al., 1965; Taylor et al., 1971).

In examining what physiological stimuli promote FFA mobilization during prolonged moderate exercise, Basu et al. (1960) exercised 10 subjects on a treadmill for a period of 60 minutes. $\dot{V}O_2$ varied from 0.8 to 1.4 liters/minute. FFA concentrations examined at 15 minute intervals showed a continuous rise above resting level. Four of the subjects whose FFA concentrations were sampled prior to 15 minutes showed an initial decrease below resting level. Havel et al. (1963), using a similar model, exercised fasted and non-fasted subjects for a period of 120 minutes. During exercise, plasma FFA concentrations increased. Fasted subjects had FFA concentrations which were 6-fold greater, and the turnover 4-fold greater than the non-fasted subjects. He (Laval et al., 1963) noted that the rate of efflux of FFA changed rapidly in relation to exercise and followed closely in time with the changes in $\dot{V}O_2$ and pulse rate. The rate of influx of FFA from stores into the plasma changed more slowly. The smaller contribution of circulating FFA

to oxidative metabolism in the non-fasted subjects appeared to result from the inhibition of the mobilization at the adipose cell by circulating carbohydrates. Friedberg et al. (1963) demonstrated the initial decrease in concentration followed by a continuous increase of FFA throughout a 40-45 minute exercise. In this experiment, 5 subjects exercised on a bicycle ergometer at one submaximal, absolute workload. The initial fall was interpreted to reflect a lag period required for mobilization factors to become fully operative. He (Friedberg et al., 1963) noted that when FFA eventually rose, mobilization factors exceeded those of utilization, resulting in higher FFA levels.

The rates of FFA mobilization and of uptake by the muscle are higher during exercise than at rest at any equivalent FFA level (Issekutz et al., 1964; Paul, 1970). Working with palmitate-1-^{14}C infusion in dogs, Issekutz et al. (1964) showed the rate of FFA uptake increase linearly with FFA concentration during rest and exercise. At rest, 20-22% of the FFA uptake was oxidized while during exercise this rose to 80-90%. In fasted resting men, 10% of the radioactivity contained in either palmitate-1-^{14}C or oleate-1-^{14}C was oxidized to $^{14}CO_2$ for the first hour after injection of the isotope (Fredrickson and Gordon, 1958). Havel et al. (1967) showed that half the FFA leaving the plasma was oxidized directly to CO_2 during vigorous muscular exercise. In evaluating the behavior of FFA, Keul (1972, p. 157) states:

> ....homogeneous behavior of the FFA level cannot be expected and that even under strictly defined conditions, there will be large variations in the amount of FFA in the blood.

Summary: Thus it has been shown that fat is available in the body as chylomicrons, TG, and FFA and that the metabolic contribution to energy production is achieved through the β-oxidation of FFA. The uptake of

FFA is dependent on mass action. The utilization by muscle mitochondria is dependent upon carnitine transfer and metabolic rate. FFA mobilization has been shown to be increased by epinephrine, norepinephrine, growth hormone, cortisol, and ACTH. Under exercise conditions at submaximal workloads, FFA levels tend to initially decrease due to differences in fractional utilization, followed by increases of 4-6 fold above resting. FFA can contribute up to 90% of the total substrate for energy production during exercise.

Lactate

During exercise, an interplay between oxidative and glycolytic metabolism takes place. Current available literature (Paul, 1975; Holloszy, 1975) demonstrates that carbohydrate and lipid as of equal importance during exercise. However, as exercise intensity increases, the importance of glycogen as an energy substrate (Pruett, 1970) also increases.

The appearance of lactate during exercise from the glycolytic breakdown of glucose or glycogen can be expressed in terms of the individual's $\dot{V}O_2$ max. This may range from 40% in unfit individuals (Davies and Harris, 1973; Davies and Harris, 1968; Pruett, 1970). With work continued beyond 10-15 minutes, blood lactate values begin to decrease (Nagle et al., 1970; Saiki et al., 1967; Wasserman et al., 1965; Wasserman et al., 1967), and in prolonged exercise blood lactate levels return to near resting values (Astrand et al., 1963; Costill, 1970; Costill et al., 1973; Karlsson, 1971; Keul et al., 1974).

The direction of change in plasma FFA concentrations during exercise is dependent upon the relative severity of the work as measured by $\dot{V}O_2$ (Pruett, 1970). The relative severity of the work also dictates whether

or not lactate is to be formed. The finding by Issekutz and Miller (1962b) that there exists an inverse correlation between the FFA and lactate levels in the blood indicated that lactate might function as a physiological inhibitor of FFA mobilization in severe exercise. They (Issekutz and Miller, 1962b) also noted that infusion of lactic acid in a resting dog decreased plasma FFA level. Subsequent studies (Miller et al., 1963; Rodahl et al., 1964; Issekutz et al., 1965a; Issekutz, 1966) showed that lactate inhibits mobilization, but not uptake of FFA (Miller et al., 1963). Pruett (1970), using healthy trained male subjects found that, for workloads up to 70-80% of $\dot{V}O_2$ max, exercise activates agents which increase plasma FFA thus increasing substrate availability as the exercise is prolonged. As exercise becomes more severe in relation to the subject's $\dot{V}O_2$ max, so that a larger proportion of energy is derived anaerobically, the mobilization of FFA is depressed for as long as blood lactate remains high. She (Pruett, 1970), concurred with the finding of Miller et al. (1963) that although mobilization is depressed, FFA uptake can still take place, supplying 10% more of the energy at work levels of 88.5% $\dot{V}O_2$ max. The fact that lactate appearance in highly trained subjects occurs at a higher percentage of the individual's $\dot{V}O_2$ max supports the finding of Paul (1970) that well-trained individuals exercising at different levels of energy expenditure will respond with larger increases of FFA mobilization at heavier workloads, and with a larger portion of the FFA mobilized being oxidized immediately. This generalization appears to be applicable to dogs as well. Issekutz et al. (1965b) showed that unfit mongrel dogs, when compared to similar but fit animals, respond with a higher lactate production over a 30-minute period. The trained animals responded with a continual rise in

FFA plasma concentration, while untrained animals showed differences in lactate and FFA response between fit and sedentary individuals working at the same workload and $\dot{V}O_2$. The sedentary subjects responded with a more rapid heart rate, a greater rise in excess lactate, and a larger fall in FFA in the first few minutes of exercise. When fit and sedentary subjects ran on an outdoor track for 90 minutes (Johnson et al., 1969), lactate response for the first 30 minutes in the sedentary group was significantly higher than in the fit group. Lactate response in the ensuing 60 minutes dropped in the untrained subjects, while the fit subjects showed a slight increase. Workload was not equivalent over the two groups as the fit subjects ran faster (16 km/hr versus 10 km/hr), and at a more constant speed than did the unfit subjects. Despite the initial increase in lactate response of the unfit subjects, they displayed a greater FFA concentration at completion of the 90 minutes. Claremont et al. (1975) exercised subjects on a bicycle at 52 to 59% $\dot{V}O_2$ max for 30 to 60 minutes in a hot (35°C) and cold (0°C) environment. Lactate produced when subjects exercised in the warm environment was 35.9 mg%, while in the cool environment 26.5 mg%. Fink et al. (1975) observed similar increases in lactate response with bicycle ergometer exercise conducted in 41°C as opposed to 9°C. Their (Fink et al., 1975) subjects exercised at 70 to 80% $\dot{V}O_2$ max three times for a period of 15 minutes each. The three exercise bouts were interspersed with 10 minute rest periods. Lactate produced in the hot environment was nearly twice that produced in the cool environment (52 vs. 29 mg%).

Boyd et al. (1974) injected D, L sodium lactate into 6 exercising normal male subjects. Exercise was performed on a bicycle ergometer at a work level mild enough to increase FFA concentrations, but not lactate

concentrations. Infusion of sodium bicarbonate and sodium chloride served as controls. Infusion of lactate decreased both FFA and glycerol levels, whereas both continued to rise with bicarbonate and saline infusions. Boyd et al (1976) points out that the physiological significance of lactate inhibition of FFA mobilization is sound in that it occurs at a time when muscle metabolism is working anaerobically and FFA cannot be utilized under such conditions.

Summary: The direction of change of plasma FFA during exercise is dependent upon the relative severity of the work as measured by $\dot{V}O_2$. Lipids can supply up to 10% of the energy necessary at workloads of 88.5% of the $\dot{V}O_2$ max. The appearance of lactate tends to inhibit FFA mobilization, but not uptake. Trained versus untrained individuals respond differently. Trained individuals respond with a larger increase of plasma FFA at heavier workloads, whereas untrained individuals show a greater rise in lactate.

Respiratory Quotient (R.Q.)

R.Q. has been established as a variable for "determining the quantity and the character of the fuels burned by the entire body" (Owen and Reichard, 1971, p. 181).

Andres (1956) studied fourteen male subjects under basal conditions as well as during forearm exercise. Simultaneous measurement of differences in a-v concentrations of oxygen, carbon dioxide, glucose, and lactate gave an R.Q. of 0.8. He noted that most of the oxygen uptake is spent in oxidation of non-carbohydrate material and also established the major non-carbohydrate material as lipid.

Participation of fat and carbohydrate in energy metabolism was examined on the basis of R.Q. during work at different intensities by

Christiansen and Hansen (1939). The effect of a normal diet on subjects' engaging in essentially aerobic exercise resulted in 50-60% of the energy being supplied by fat. If work up to 3 hours in duration was used, fats supplied up to 70% of the energy. The energy supplied by lipids increased to 99% if a high-fat diet preceeded the exercise bout for several days. However, the ability to carry on this work decreased markedly. A carbohydrate-rich diet initially decreased the contribution of fat to as low as 25% and, as exercise continued, this increased to 60%.

Unlike Christiansen and Hansen (1939) who obtained R.Q. values of around 0.7 strictly on a high-fat diet, similar values were obtained by Issekutz et al. (1963b) regardless of the diet. In this study, the interaction of fats and carbohydrates in energy expenditure during light exercise (300 kpm/min) on a bicycle ergometer was studied in 6 healthy men, 17 to 24 years of age. Actual carbohydrate intake rather than the amount of fat in the diet was concluded as the decisive factor in determining whether fatty acids or glucose were the preferred fuel.

R.Q., during the first few minutes of exercise, is a function of oxygen supply, or lack of it, rather than a function of the fuel utilized (Issekutz et al., 1962b). However, dietary conditions as well as formation of lactate may mask the metabolic R.Q.

When Hermansen et al. (1967) exercised subjects at workloads corresponding to 29, 53, and 79% of their $\dot{V}O_2$ max, the R.Q.'s were 0.87, 0.90, and 0.93 respectively, indicating the taking over by carbohydrate metabolism with increased energy requirement.

<u>Summary</u>: Respiratory R.Q. may be used to estimate the type of fuel being used, however, diet, and the formation of lactate may mask the true metabolic R.Q.

Deep Body Temperature

Based on Nielsen's paper (1938), Saltin (1972, p. 57) states:

> ...almost all of his conclusions are still valid and little new information on body temperature regulation has been gathered since then.

Deep body temperature reaches a steady state within 20 minutes and no further increase is seen after that time (Saltin and Hermansen, 1966; Nielsen and Nielsen, 1962), even if the work is continued for 4 hours (Nielsen, 1938). Deep body temperature is believed to be related not to the actual workload, but to the percentage of $\dot{V}O_2$ max that the subject works (Saltin, 1972). He (Saltin, 1972) states also that one always achieves the same deep body temperature for a certain relative workload within environmental temperature ranges of +5°C to +30°C. Claremont et al. (1975) as well as other investigators (Robinson et al., 1963; Lind, 1963; Pugh, 1967) have demonstrated that:

> ...outside this environmental-independent zone, temperature regulating processes in extremely hot or cold ambient conditions become incapable of providing sufficient output to maintain thermal balance.
> (Claremont et al., 1975, p. 150)

Studies on the effect of external heating are not uncommon (Sancetta et al., 1958; Bell et al., 1965; Carlesten et al., 1961; Goldman et al., 1965; Eisalo, 1956; DeVries et al., 1960), however, only a few to date have examined FFA mobilization under thermal stress (Taggart et al., 1972; Britton et al., 1974; Eddy et al., 1976). Taggart et al. (1972) examined the effect of a short exposure of intense heat of a sauna bath on the electrocardiogram of normal and coronary heart disease subjects. Plasma catacholamines, FFA, and triglyceride concentrations were measured. No significant changes in plasma FFA occured

in both groups of subjects after exposure to 10 minutes of sauna heat. Britton et al. (1974) studied the adrenergic, coagulation, and fibrinolytic responses of seven male volunteers to heat exposure in a sauna. Insignificant increases in FFA were observed after exposures of 10 and 15 minutes.

The findings of Britton et al. (1974) and Taggert et al. (1972) are in conflict with Eddy et al (1976) who observed an increase in FFA levels when subjects were exposed to increasing heat in a climatic chamber. In this study (Eddy et al., 1976) the combination of thermal stress and dehydration were investigated as possible factors in elevation of FFA levels. Five clothed male subjects entered a climatic chamber at room temperature after which the chamber's temperature was elevated to a mean of 70°C. The subjects remained in the chamber for an average of 58 minutes until a deep body temperature elevation of 1.4°C was achieved. Blood samples for FFA analysis were withdrawn at two points (0.7° and 1.4°C rise in body temperature) during the heat exposure. Weight loss was recorded and the subjects, after a week's rest, repeated the same procedure. However, on the second occasion fluids were replenished in the amount of the weight loss seen in the initial heat exposure. A significant ($p < 0.05$) difference in FFA concentration was seen between the two trials at body temperatures 1.4°C above rest. Resting FFA levels were 0.5 uEq/ml for both trials. For subjects who dehydrated, FFA levels rose to 1.4 uEq/ml while with rehydration FFA levels rose only to 0.7 uEq/ml. Lactates which were also measured did not change from rest values of 8.4 to 10.6 mg%.

Summary: During exercise deep body temperature is elevated and remains steady at points relative to percentage of $\dot{V}O_2$ max at which the

individual is working. There is conflicting evidence as to whether or not increases in deep body temperature affect FFA levels. While rapid elevation of deep body temperature (10 to 15 minutes) using a sauna appears to have no significant effect on plasma FFA levels, a more prolonged exposure (58 minutes) in a climatic chamber accompanied by dehydration will elevate FFA levels.

Warm-up Effects

Increased heat production associated with muscular exercise leads to a rapid temperature rise in the working muscles and a more gradual increase in body core temperature (Astrand and Rodahl, 1970). Theoretical calculations, made from available data on muscle blood flow and capacity of the blood to transport heat, suggests the difference between exercising muscle and deep body temperature to be between 0.5 and 0.7°C (Grimby, 1967).

Preliminary exercise as a preparation prior to physical work attempts to produce optimum performance through psychological as well as physiological factors. Barcroft and King (1910-11) noted the dissociation of oxygen from hemoglobin is more complete at higher muscle temperatures, thus enhancing oxygen supply during work.

Stuart (1974) evaluated the effect of increases in deep body temperature of 0.5°C on a speed and endurance criterion test. Preliminary warm-up at 50% of the subjects' predicted $\dot{V}O_2$ max based on a heart rate of 135 showed superior performance in speed and endurance. If 75% of the subjects' predicted $\dot{V}O_2$ max was used, no such increase was found. Asmussen and Boje (1946) showed in 4 trained subjects that an increase of 0.8°C rectal temperature during a 30-minute warm-up improved both short and long term performance on a bicycle ergometer. The Asmussen

and Boje study (1946) concluded that "a greater amount of work was performed and a greater tension developed when subjects were warmed up." The increased ability to do work was related to muscle temperature increase. Reasons given were that $\dot{V}O_2$ at a given workload decreased and $\dot{V}O_2$ max increased with warm-up.

Preliminary treadmill exercise warm-up at 40 to 50% $\dot{V}O_2$ max for 15 minutes was used by Horstman et al. (1971) to evaluate warm-up effects on moderate (40 to 50% max) and heavy (80 to 90% $\dot{V}O_2$ max) work tasks. Both the moderate and heavy work tasks lasted for 15 minutes, with the moderate task being a continuation of the warm-up exercise load. For subjects who walked 30 minutes at the 40 to 50% $\dot{V}O_2$ max load, FFA concentrations decreased slightly over the first 15 minutes (0.5 to 0.474 uEq/ml), then increased to basal levels (0.529 uEq/ml) over the next 15 minutes. The latter 15 minutes were considered the moderate work task. If the heavy work task (80 to 90% $\dot{V}0_2$ max) was instituted for the last 15 minutes, the FFA concentration continued to decrease to a level significantly lower (p 0.01) than the value obtained when the moderate workload was used.

Astrand and Rodahl (1970, p. 524) sum up the benefits of increasing the deep body temperature through warm-up stating:

> ...metabolic processes in the cell can proceed at higher rate, since these processes are temperature-dependent.

Through such arguments, warm-up has been deemed essential for optimum performance. Martin et al. (1975, p. 146), however, states that "actual physiological data on the question has been indeterminate." In assessing the effects of warm-up on both aerobic and anerobic energy transformations in treatmill exercise of 1.5 and 5 minutes durations, they (Martin et al.,

1975) showed an 11% increase in heart rate and an 8% increase in $\dot{V}O_2$ when warm-up was used. Running following warm-up resulted in 25% lower lactate production as well as 3 to 4°C higher gastrocnemius muscle temperature.

APPENDIX B

TABLE 3

PAIRED 'T' - TESTS:

SELECTED PRE- AND POST-EXPERIMENTAL VARIABLES

			VARIABLES			
			workload (kpm/min)	heart rate (bpm)	$\dot{V}O_2$ max (L/min)	weight (kg)
MAXIMAL TEST	initial	MEAN	1909.1	189.8	4.02	75.32
		$S_{\bar{E}}$	64.25	1.96	0.107	2.31
	final	MEAN	1936.4	189.7	3.99	74.64
		$S_{\bar{E}}$	58.81	2.56	0.17	2.15
		T	0.626	-0.894	-2.008	1.052
		P	0.547	0.395	0.076	0.32

TABLE 4

ANALYSIS OF VARIANCE FOR PLASMA FREE FATTY ACIDS

source of variation	SS	DF	MS	F	P
between subjects	4.306	109			
'A' main effects	0.506	9	0.056	1.48	0.166
subjects between groups	3.800	100	0.038		
within subjects	1.200	220			
'B' main effects	0.072	2	0.036	6.531	0.0018
A * B interaction	0.078	18	0.004	0.780	0.7221
'B' x subjects within groups	1.110	200	0.006		

TABLE 5

NEWMAN-KEULS TEST FOR COMPARISON OF PLASMA FREE FATTY ACIDS MEANS FOR CONTROL, EXERCISE, AND SAUNA TREATMENT CONDITIONS DURING THE RECOVERY 15-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
mean	0.692	0.786	0.746
$S_{\bar{E}}$	0.036	0.058	0.052
CONTROL		*	
EXERCISE			
SAUNA			

* significant at $p<0.05$

TABLE 6

ANALYSIS OF VARIANCE FOR LACTATE

source of variation	SS	DF	MS	F	P
between subjects	23618.813	109			
'A' main effects	11722.484	9	1302.498	10.949	0.0
subjects within groups	11896.313	100	118.963		
within subjects	4751.875	220			
'B' main effects	764.157	2	382.078	22.244	0.0
'A * B' interaction	552.407	18	30.689	1.787	0.029
'B' x subject within groups	3435.375	200	17.177		

TABLE 7

NEWMAN-KEULS TEST FOR COMPARISON OF LACTATE MEANS FOR THE CONTROL TREATMENT CONDITION DURING REST, EXERCISE, AND RECOVERY

		SAMPLING INTERVALS									
MEAN ($S_{\bar{E}}$)		R_1	R_2	PT	5	10	15	20	25	30	REC
9.06 (0.5)	R_1				*	*	*	*	*	*	
8.9 (0.84)	R_2				*	*	*	*	*	*	
8.36 (0.35)	PT				*	*	*	*	*	*	
18.17 (2.15)	5										
23.02 (2.73)	10										*
23.36 (3.45)	15										*
22.75 (3.43)	20										*
22.81 (3.40)	25										*
22.65 (3.48)	30										*
12.36 (1.70)	REC										

* significant at $p<0.05$

TABLE 8

NEWMAN-KEULS TEST FOR COMPARISON OF LACTATE MEANS FOR THE EXERCISE TREATMENT CONDITION DURING REST, EXERCISE, AND RECOVERY

			SAMPLING INTERVALS									
			R_1	R_2	PT	5	10	15	20	25	30	REC
MEAN	($S_{\bar{E}}$)											
8.45	(0.71)	R_1									*	
8.49	(0.79)	R_2									*	
8.66	(0.67)	PT									*	
16.94	(1.47)	5										
17.96	(1.72)	10										
18.08	(1.91)	15										
17.85	(1.79)	20										
18.01	(1.83)	25										
18.37	(2.09)	30										
10.33	(0.77)	REC										

* significant at $p<0.05$

TABLE 9

NEWMAN-KEULS TEST FOR COMPARISON OF LACTATE MEANS FOR THE SAUNA TREATMENT CONDITION DURING REST, EXERCISE, AND RECOVERY

			SAMPLING INTERVALS									
			R_1	R_2	PT	5	10	15	20	25	30	REC
MEAN	($S_{\bar{E}}$)											
8.13	(0.67)	R_1				*	*	*	*	*	*	
7.76	(0.56)	R_2				*	*	*	*	*	*	
9.25	(0.61)	PT				*	*	*	*	*	*	
18.84	(1.70)	5										*
22.90	(2.47)	10										*
23.97	(2.77)	15										*
24.42	(3.04)	20										*
25.51	(3.54)	25										*
25.20	(3.61)	30										*
12.31	(1.24)	REC										

* significant at $p<0.05$

TABLE 10

NEWMAN-KEULS TEST FOR COMPARISON OF LACTATE MEANS FOR CONTROL, EXERCISE, AND SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-10-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	23.02	17.96	22.90
$(S_{\bar{E}})$	2.73	1.72	2.47
CONTROL		*	
EXERCISE			*
SAUNA			

* significant at $p<0.05$

TABLE 11

NEWMAN-KEULS TEST FOR COMPARISON OF LACTATE MEANS FOR CONTROL, EXERCISE, AND SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-15-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	23.36	18.08	23.97
$(S_{\bar{E}})$	3.45	1.9	2.77
CONTROL		*	
EXERCISE			*
SAUNA			

* significant at $p<0.05$

TABLE 12

NEWMAN-KEULS TEST FOR COMPARISON OF LACTATE MEANS FOR CONTROL, EXERCISE, AND SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-20-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	22.75	17.85	24.42
$(S_{\bar{E}})$	3.43	1.79	3.04
CONTROL		*	
EXERCISE			*
SAUNA			

* significant at $p<0.05$

TABLE 13

NEWMAN-KEULS TEST FOR COMPARISON OF LACTATE MEANS FOR CONTROL, EXERCISE AND SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-25-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	22.31	18.01	25.51
$(S_{\bar{E}})$	3.40	1.83	3.54
CONTROL		*	
EXERCISE			*
SAUNA			

* significant at $p<0.05$

TABLE 14

NEWMAN-KEULS TEST FOR COMPARISON OF LACTATE MEANS FOR CONTROL, EXERCISE, AND SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-30-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	22.65	18.37	25.20
$(S_{\bar{E}})$	3.48	2.09	3.61
CONTROL		*	
EXERCISE			*
SAUNA			

* significant at $p<0.05$

TABLE 15

ANALYSIS OF VARIANCE FOR RESPIRATORY QUOTIENT

source of variation	SS	DF	MS	F	P
between subjects	1.117	65			
'A' main effects	0.049	5	0.010	0.553	0.736
subjects within groups	1.068	60	0.018		
within subjects	1.171	132			
'B' main effects	0.103	2	0.052	5.894	0.004
A * B interaction	0.015	10	0.002	0.176	0.998
'B' x subjects within groups	1.053	120	0.009		

TABLE 16

ANALYSIS OF VARIANCE FOR DEEP BODY TEMPERATURE

source of variation	SS	DF	MS	F	P
between subjects	54.313	109	5.553		
'A' main effects	49.973	9	5.553	128.754	0.0
subjects within groups	4.313	100	0.043		
within subjects	25.938	220			
'B' main effects	7.949	2	3.975	56.030	0.0
'A * B' interaction	4.211	18	0.234	3.298	0.0
'B' x subject within groups	14.188	200	0.071		

TABLE 17

NEWMAN-KEULS TEST FOR THE COMPARISON OF DEEP BODY TEMPERATURE FOR THE CONTROL TREATMENT CONDITION DURING REST, EXERCISE AND RECOVERY

			SAMPLING INTERVALS									
			R_1	R_2	PT	5	10	15	20	25	30	REC
mean	($S_{\bar{E}}$)											
36.76	(0.06)	R_1						*	*	*	*	*
36.76	(0.06)	R_2						*	*	*	*	*
36.78	(0.06)	PT						*	*	*	*	*
36.85	(0.06)	5						*	*	*	*	*
37.06	(0.06)	10							*	*	*	*
37.21	(0.12)	15							*	*	*	*
37.45	(0.05)	20									*	
37.63	(0.06)	25										
37.77	(0.07)	30										*
37.50	(0.08)	REC										

* significant at $p<0.05$

TABLE 18

NEWMAN-KEULS TEST FOR THE COMPARISON OF DEEP BODY TEMPERATURE FOR THE EXERCISE TREATMENT CONDITION DURING REST, EXERCISE AND RECOVERY

			SAMPLING INTERVALS									
			R_1	R_2	PT	5	10	15	20	25	30	REC
mean	$(S_{\bar{E}})$											
36.77	(0.06)	R_1			*	*	*	*	*	*	*	*
36.79	(0.06)	R_2			*	*	*	*	*	*	*	*
37.29	(0.09)	PT						*	*	*	*	
37.34	(0.07)	5							*	*	*	
37.48	(0.07)	10								*	*	
37.60	(0.09)	15									*	
37.71	(0.07)	20										*
37.81	(0.08)	25										*
37.90	(0.08)	30										*
37.35	(0.15)	REC										

* significant at $p<0.05$

TABLE 19

NEWMAN-KEULS FOR THE COMPARISON OF DEEP BODY TEMPERATURE FOR THE SAUNA TREATMENT CONDITION DURING REST, EXERCISE, AND THE RECOVERY

			SAMPLING INTERVALS									
			R_1	R_2	PT	5	10	15	20	25	30	REC
mean	$(S_{\bar{E}})$											
36.76	(0.05)	R_1			*	*	*	*	*	*	*	*
36.77	(0.05)	R_2			*	*	*	*	*	*	*	*
37.09	(0.05)	PT					*	*	*	*	*	*
37.48	(0.06)	5						*	*	*	*	
37.66	(0.06)	10							*	*	*	
37.80	(0.07)	15								*	*	
37.94	(0.06)	20										*
38.06	(0.06)	25										*
38.16	(0.07)	30										*
37.61	(0.09)	REC										

* significant at $p<0.05$

TABLE 20

NEWMAN-KEULS TEST FOR COMPARISON OF DEEP BODY TEMPERATURE MEANS FOR CONTROL, EXERCISE, AND SAUNA TREATMENT CONDITIONS DURING THE POST-TREATMENT SAMPLE

	CONTROL	EXERCISE	SAUNA
means	36.78	37.29	37.29
$(S_{\bar{E}})$	0.06	0.06	0.05
CONTROL		*	*
EXERCISE			
SAUNA			

* significant at $p<0.05$

TABLE 21

NEWMAN-KEULS TEST FOR COMPARISON OF DEEP BODY TEMPERATURE MEANS FOR CONTROL, EXERCISE AND SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-5-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	36.85	37.34	37.48
$(S_{\bar{E}})$	0.06	0.07	0.06
CONTROL		*	*
EXERCISE			
SAUNA			

* significant at $p<0.05$

TABLE 22

NEWMAN-KEULS TEST FOR COMPARISON OF DEEP BODY TEMPERATURE MEANS FOR CONTROL, EXERCISE ANS SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-10-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	37.06	37.47	37.66
($S_{\bar{E}}$)	0.06	0.07	0.06
CONTROL		*	*
EXERCISE			
SAUNA			

* significant at $p<0.05$

TABLE 23

NEWMAN-KEULS TEST FOR COMPARISON OF DEEP BODY TEMPERATURE MEANS FOR CONTROL, EXERCISE AND SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-15-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	37.21	37.60	37.80
($S_{\bar{E}}$)	0.12	0.07	0.07
CONTROL		*	*
EXERCISE			
SAUNA			

* significant at $p<0.05$

TABLE 24

NEWMAN-KEULS TEST FOR COMPARISON OF DEEP BODY TEMPERATURE MEANS FOR CONTROL, EXERCISE, ANS SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-20-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	37.45	37.71	37.94
$(S_{\bar{E}})$	0.05	0.07	0.06
CONTROL		*	*
EXERCISE			*
SAUNA			

* significant at $p<0.05$

TABLE 25

NEWMAN-KEULS TEST FOR COMPARISON OF DEEP BODY TEMPERATURE MEANS FOR CONTROL, EXERCISE ANS SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-25-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	37.63	37.81	38.06
$(S_{\bar{E}})$	0.06	0.08	0.06
CONTROL			*
EXERCISE			*
SAUNA			

* significant at $p<0.05$

TABLE 26

NEWMAN-KEULS TEST FOR COMPARISON OF DEEP BODY TEMPERATURE MEANS FOR CONTROL, EXERCISE ANS SAUNA TREATMENT CONDITIONS DURING THE EXERCISE-30-MINUTE SAMPLE

	CONTROL	EXERCISE	SAUNA
means	37.77	37.90	38.16
$(S_{\bar{E}})$	0.07	0.08	0.07
CONTROL			*
EXERCISE			*
SAUNA			

* significant at $p < 0.05$

TABLE 27

ANALYSIS OF VARIANCE FOR OXYGEN CONSUMPTION

source of variation	SS	DF	MS	F	P
between subjects	9.670	65			
'A' main effects	1.049	5	0.210	1.460	0.216
subjects within groups	8.621	60	0.144		
within subjects	8.058	132			
'B' main effects	0.241	2	0.121	1.862	0.160
'A * B' interaction	0.046	10	0.005	0.070	0.999
'B' s subject within groups	7.771	120	0.065		